

Asthma And Damp Housing

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Thesis submitted for the degree of

Doctor of Medicine

University of Edinburgh

1999



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Declaration

I declare that I have made a substantial contribution to the work described in this thesis. I contributed to the design of the study and organised the recruitment of all the asthmatic and control subjects. The interview based questionnaires and dampness surveys were conducted by professionals in these fields. All of the data and statistical analysis was performed by myself. I confirm that this thesis was written entirely by myself and has not been submitted for any other degree, diploma or professional qualification.

Date 24th February 1999

Acknowledgements

I would like to thank the following:

Dr Tony Fennerty for his ideas, support and correction of the text of the thesis. Dr Robert Monie and Claudia Martin for their help and support. Gail Vasey, Catherine Gilles, Anne Bryce and Laura Cunningham for conducting the interview based questionnaires. George McGill of Healthy homes Ltd., Glasgow for conducting the dampness surveys. My wife Christine for her encouragement and support whilst writing the thesis. The Chest, Heart and Stroke Association Scotland for financing the project.

Abstract

Background

Several epidemiological studies have reported a higher prevalence of respiratory symptoms in subjects living in damp housing, but links with specific respiratory diseases such as asthma have not been satisfactorily established. The aims of this thesis were to determine if there is an association between damp housing and asthma and to investigate whether damp housing adversely influences asthma severity.

Methods

102 subjects with physician diagnosed asthma attending a hospital out-patient clinic and 196 age and sex matched controls completed an interview based questionnaire giving details on health, social and behavioural information. 222 (75%) agreed to have their dwelling surveyed for damp and mould growth by an independent surveyor who recorded indoor room temperatures, relative humidity, dampness measurements and presence of visible mould growth using standardised, reproducible methodology. The prevalence of both self-reported and observed damp and mould growth in the homes of the asthmatic and control subjects were compared. An asthma severity score was calculated for each asthma patient such that disease severity could be correlated with the severity of damp in the dwelling.

Results

Asthmatic subjects reported more damp in both their current (Odds Ratio 4.1, 95%CI 2.3 to 7.6) and previous (Odds Ratio 1.9, 95%CI 1.1 to 3.2) dwellings than control subjects. The surveyor confirmed 112 (51%) dwellings to have evidence of damp and 57 (26%) evidence of visible mould growth. Dampness was detected in 58/90 (64%) dwellings of asthmatic subjects compared with 54/132 (41%) dwellings of control subjects (Odds Ratio 2.62, 95%CI 1.50 to 4.55). This association could not be explained by potential bias in study design and persisted after controlling for socio-economic and other confounding variables (adjusted odds ratio 3.03, 95% CI 1.65 to 5.57).

Asthma severity scores correlated statistically with measures of total damp ($r=0.30$, $p=0.006$) and visible mould growth ($r=0.23$, $p=0.035$) in the dwelling. Patients living in homes with evidence of damp had a lower FEV1 (mean difference 10%, 95% CI 1.0 to 20) and a lower FEV1/FVC ratio (mean difference 5.4%, 95% CI -0.1 to 10.9) than patients living in dry dwellings. These associations persisted after controlling for unemployment, household income and cigarette smoking.

There was no association between a subject's atopic status or sensitivity to house dust mite and the presence of damp or mould in the home. Asthmatic subjects living in dwellings with extensive mould were more likely to be mould sensitive on skin prick testing ($p=0.03$)

Conclusions

Asthma is significantly associated with living in damp housing. Measures of asthma severity, disability and airflow obstruction are higher in patients living in damp, mouldy dwellings. Effective measures to reduce the risk of damp and condensation occurring in dwellings are required to be incorporated into future housing design. This may improve the quality of life of asthma sufferers and their families.

Chapter 1

Introduction

Section I

Asthma definition and aetiology

1.1 Definition of asthma

Asthma is a common chronic respiratory illness affecting all ages of the population. It has been recognised as a distinct disease for many hundreds of years, but despite the passage of time, still defies precise definition even today. One of the first attempts to propose a standard definition of asthma was made by the American Thoracic Society in 1962 (1). It stated that "the disease is characterised by the presence of increased hyper-responsiveness and wide-spread airway narrowing that can change in severity either spontaneously or with therapy". Despite our increased understanding of the pathology of asthma over the last thirty years, further carefully worded statements still fail to offer a precise definition (2-4). It is agreed the diagnosis should be made by recognition of a pattern of characteristic symptoms and is best confirmed by evidence of variable or reversible airflow obstruction.

Descriptive definitions of asthma such as those cited above do not translate well for use in epidemiological studies. Here, the lack of an objective gold standard for asthma diagnosis is particularly troublesome. Subjects with mild asthma may have no observed evidence of airflow obstruction and may fail to show either significant variability on serial peak flow rate monitoring or evidence of bronchial hyper-responsiveness, resulting in their being under-represented in asthma surveys. Most subjects with moderate to severe asthma will have demonstrable bronchial hyper-responsiveness either to non-specific stimuli such as exercise or to chemicals such as histamine or metacholine. However, approximately ten percent of the general population also have increased bronchial hyperreactivity but have no symptoms of asthma (5-6). Increased airway responsiveness is also a feature of other respiratory diseases including chronic obstructive pulmonary disease (7). Bronchial hyper-responsiveness is therefore one of several factors which may indicate the presence of asthma, but alone cannot be used to diagnose the condition. A compromise of the presence of bronchial hyper-responsiveness and wheeze in the last twelve months has been recently proposed as an epidemiological definition for asthma (8), which if adopted may allow more direct comparisons between different studies.

An alternative epidemiological approach, undertaken by some authors, is to simply report on cases of physician diagnosed asthma. Such a definition is likely to select subjects with more severe disease, which led to their seeking medical help and being labelled and treated as having asthma. Different attitudes and access to health care may therefore influence the selection of asthmatic subjects when using such a definition in epidemiological studies. Despite these limitations, a definition

of physician diagnosed asthma is frequently used in studies of case-control design and was the definition of asthma chosen for the purpose of this thesis. This would give the advantage of including only unequivocal cases of asthma.

1.2 Asthma genetics

One of the first studies of the heredity of asthma and hay fever was performed by Cooke *et al* in 1916 (9). They emphasised the familial nature of the disease and provided evidence for genetic predisposition. It is now recognised that atopic diseases such as asthma are complex genetic disorders that do not follow a simple Mendelian pattern. Rather, their development is believed to depend upon interactions between several genes, at least one of which is known to be HLA linked, and exposure to certain environmental factors.

The lack of a precise definition and the influence of the environment on disease expression has hindered the search for the genetic cause of asthma. For these reasons, genetic linkage studies have tended to focus on possible sites controlling bronchial hyperresponsiveness and IgE production, both of which are easily measured and thought to be important in the development of asthma.

Familial studies have provided evidence of a genetic influence on bronchial hyperreactivity (BHR), as Hopp *et al* found a higher concordance of BHR in monozygotic than in dizygotic twins (10). The same author also reported that, in a population study, there was a greater prevalence of BHR in non-asthmatic subjects with a history of atopy than in those without a history of atopy (11).

The ability to over-produce IgE when exposed to specific antigens is also genetically inherited (12). The Tuscon epidemiological study of airways obstructive diseases found the single most significant predictor of childhood asthma to be an interaction between asthma in either parent and the maternal IgE level (13). Genetic linkage studies have identified a relationship between atopy and chromosome 11q13. Variants of the beta-subunit of the high affinity IgE receptor are encoded in this region and an amino acid substitution of Leucine for Isoleucine at position 183 has a strong association with atopy by maternal descent (14). Other studies have found linkage of both total serum IgE and bronchial hyper-responsiveness to markers on chromosome 5q (15). This region contains genes regulating a large number of factors

important in the inflammatory process which is characteristic of allergy and asthma, but further work is required to clarify the specific genetic abnormality predisposing to the development of asthma.

1.3 Asthma and the environment

Although the predisposition for asthma is inherited, there is clear evidence that the principal determinant of asthma is environmental. Surveys from developing countries have shown a low prevalence of bronchial hyper-responsiveness in the population living in poor rural areas, but this low prevalence increases rapidly as populations move to urban, more affluent areas (16-17). A dramatic increase in adult asthma in the South Fore region of Papua New Guinea (from 0.15 to 7.3%) was related to the introduction of blankets, which became infested with house dust mites (18).

Further evidence supporting the role of the environment in the development of asthma has been the apparent increase in asthma prevalence over the past three decades. Indirect evidence is provided by an increase in the proportion of general practitioners' patients attending their doctors with asthma or hay fever in the UK (19). This however may be partly due to increased disease awareness or change in diagnostic labelling that have occurred during this time period. More direct evidence arises from studies which have measured the prevalence of asthma in the same population at two points in time. There are several such studies from Britain (20-22), New Zealand (23), Australia (24) and the USA (25), all showing an increase in the prevalence of wheezing and asthma in children over time.

The increase in the prevalence of asthma and atopic symptoms is therefore likely to be attributable to some form of adverse environmental changes, as they have occurred too rapidly to be explained by a change in the gene pool resulting in an increased prevalence of genetically predisposed subjects in the population. The environmental factors which have been postulated to explain the increasing prevalence of asthma in affluent countries include greater exposure to various air pollutants, smaller family size, changed dietary factors, and increased exposure to indoor allergens. Each will be discussed briefly in turn.

1.3.1 Atmospheric Pollution

An increasing degree of attention has been focused on air pollution in industrialised countries and its influence on respiratory disorders. With the recent political changes in Eastern Europe, several studies have been able to compare the prevalence of respiratory disorders in subjects living in Eastern European cities which are heavily polluted with sulphur dioxide and subjects living in Western European cities exposed to higher concentrations of pollutants such as nitrogen dioxide and ozone. Results have shown a higher prevalence of chronic bronchitis in Eastern European citizens and a higher prevalence of atopy in Western European citizens (26-27).

Several studies have shown that living on or near busy roads has a detrimental effect on respiratory health of children (28-31). Symptoms of chronic cough, chronic phlegm, wheeze and shortness of breath are commoner in these children (odds ratios range between 1.2 to 2.8). In one of these studies conducted in Munich, Germany (28), pulmonary function decreased in primary school children as traffic on the main road in the school district increased. Attendances at accident and emergency departments for acute childhood wheezy episodes in London, were related to fluctuations in atmospheric ozone and sulphur dioxide concentrations (32). Daily variations in atmospheric ozone concentration in London may adversely effect daily mortality from respiratory diseases (33). Confirmation of an increased prevalence of asthma in subjects living near busy roads is however lacking (34).

Air pollution may be responsible for some of the increase in the prevalence of allergic disease. In Japan, cases of pollinosis were rare 50 years ago, but over recent years there has been a dramatic increase in this condition. Ishizaki and colleagues (35) found that people living along-side busy main roads lined with cedar trees had a higher incidence of allergic rhino-conjunctivitis (13.2%) compared to residents in the cedar forest exposed to less intense traffic (5.1%), despite similar pollen levels in both areas.

In Switzerland Gassner and co-workers (36) found the prevalence of allergic sensitisation to grass pollens has increased in parallel with an increase in emission of photo-oxidants in exhaust fumes from motor traffic. These workers postulated that exposure to high levels of nitrogen dioxide and ozone may reduce bronchocilliary clearance and increase airway permeability, potentiating allergen exposure (37). In San Diego USA, personal ozone exposure was independently associated with asthma severity (38). Further evidence that exposure to air pollutants may

potentiate airway responsiveness to inhaled allergens was provided by Devalia (39). They showed that exposure to sulphur dioxide and nitrogen dioxide in concentrations encountered in heavy traffic enhanced the airway response to inhaled allergen.

In Cardiff, South Wales, the rise in the prevalence of allergic diseases such as hay fever between 1973 and 1988 was investigated (40). During this time annual grass pollen measurements remained remarkably constant, suggesting no change in relevant allergen exposure. Outdoor pollution measurements showed a reduction in the levels of sulphur dioxide and particulate matter over the same period, but levels of ozone or nitrogen dioxide were not available. However, there is no evidence of a rise in ozone levels in Britain over the last twenty years (41), suggesting the rise in allergic disease is unlikely to be entirely due to concomitant exposure to allergens and outdoor air pollution.

1.3.2 Environmental Tobacco Smoke

Prenatal exposure to tobacco smoking has been implicated in the development of allergic disease and impaired lung function in infancy. Conflicting evidence suggests that maternal smoking during pregnancy increases levels of IgE in cord blood and the likelihood of developing allergic disease later in infancy (42-44). Maternal smoking during pregnancy is also associated with significant reductions in forced expiratory flow rates in young infants (45).

Environmental tobacco smoke is one of the commonest indoor air pollutants and passive smoking has been implicated in the development of respiratory symptoms (46-47). There is now convincing evidence that daily exposure to environmental tobacco smoke results in an increased risk of developing airway diseases such as wheezy bronchitis (48), asthma (49-50) and bronchial hyperreactivity (51) in atopic and non-atopic children (odds ratios ranging from 1.5 to 2.5). There may also be a synergistic effect between exposure to environmental tobacco smoke and other risk factors for respiratory symptoms such as damp housing (51-52).

Asthmatic children exposed to environmental tobacco smoke have an increased morbidity, with an increased need for medical treatment and asthma medication (53). Further, a reduction in exposure to environmental tobacco smoke results in a decrease in disease severity in asthmatic children (54).

1.3.3 Family Size

Affluent countries tend to have smaller family sizes. Children without siblings are at greater risk of developing atopic illness and symptoms consistent with asthma (55-57). The greater the number of siblings at home, the more likely a child is to experience common infections early in life. Early childhood viral infections may protect against allergic disease by influencing T lymphocyte development (58). Evidence to support this hypothesis is currently weak (59-60). Most of the epidemiological data are retrospective with few clues as to which organisms may be protective. One study in Guinea-Bissau suggested infection with the measles virus protects against atopy (61), whilst a cross sectional study of Italian military students found a lower prevalence of atopy in subjects with antibodies to hepatitis A virus (62). Prospective studies to determine age of infection and type of organism and the role of vaccination are required to explore this hypothesis.

1.3.4 Dietary factors

During the past twenty years diet has changed considerably, particularly in affluent countries and this may have contributed to the increase in prevalence of asthma observed in children over the same period.

Infants who are breast-fed for a prolonged period have less allergic disease in early childhood (63-64) and a lower prevalence of asthma (65). The mechanism is unclear, but breast milk is likely to protect against some infections and prevent early exposure to foreign proteins in cow's milk. A decrease in the number of breast fed infants, or a reduction in the duration of breast feeding could therefore influence asthma prevalence.

Two other dietary factors which have been found to be independently associated with asthma are increased salt intake, which is thought to increase asthma severity and regular fish intake which may protect against airway inflammation.

Increased intake of salt in the diet has been associated with an increase risk of asthma (66) and in Italy was associated with respiratory symptoms and airway responsiveness in boys aged 9 - 16 years (67). In Kenyan school children, increased dietary salt intake was found to be an independent risk factor for asthma (68). However, clinical studies of asthmatic subjects given low salt diets have failed to demonstrate any change in asthma morbidity.

In Australia, eating fish on a regular basis appears to protect children from developing bronchial hyper-responsiveness (69) and asthma (70). Fish oils may have an anti-inflammatory role (71), which may lead to a reduction in bronchial hyperresponsiveness and expression of asthma symptoms. Clinical studies have again failed to show an improvement in asthma morbidity in subjects taking dietary fish oil supplements (72).

It is postulated there has been a reduction in dietary antioxidants, particularly vitamins C and E, in the Westernised diet (73). These vitamins may play a role in host defence against oxidative lung damage (74), but it is not known whether they have a protective role against the development of asthma.

1.3.5 Indoor Allergen Exposure

Indoor allergen exposure is considered the most important risk factor for the development and expression of allergic diseases such as asthma, rhinitis and atopic eczema. Allergens from house dust mites, pet dander, insects such as the cockroach, and moulds have all been implicated (75). The development of monoclonal antibody immunoassays to these allergens has allowed estimates of exposure within dwellings and risk levels for the development of disease.

Exposure to airborne allergens invariably occurs in infancy, if not in utero. The genetic potential to manifest allergic reactivity to these allergens, may depend upon the infant's T-cell systems' response to these initial contacts. Active immunological recognition of these environmental allergens is likely to be a normal part of the development of the immune system in early childhood (76), but in genetically predisposed infants, this allergen exposure may also be critical in the development of asthma. Month of birth studies relating aeroallergen exposure in infancy to the development of allergy and asthma support this hypothesis (77-80).

The most important allergens in the development of asthma are those derived from the house dust mite, the most prevalent species in Britain being *Dermatophagoides pteronyssinus* (81). Young *et al* conducted a retrospective study of the exposure to house dust mite allergen (*Der p* I) in young house dust mite sensitive children and their siblings (82). The house dust mite atopic children had significantly higher levels of *Der p* I in their bedding compared to their non-house dust mite sensitive atopic

siblings. They concluded that the magnitude of exposure to house dust mite allergen is important in determining specific IgE responses in children with a comparable genetic predisposition to atopy.

Sporik *et al* showed that children with house dust mite sensitivity had an increased exposure to this allergen in their homes at one year of age (83). A stronger association with increased exposure was found in a retrospective study of asthmatic children, in which sensitisation was related to the level of airborne *Der p* I estimated to have been present around the time of birth (80). They also reported that of 82 children admitted to hospital with an exacerbation of asthma, the majority were exposed to high levels of house dust mite allergen and were house dust mite sensitive (84). Further, continued exposure to higher concentrations of mite allergen were associated with a risk of re-admission.

Other studies have confirmed that exposure to concentrations of more than 2µg/g of Group I mite allergens is a risk factor for sensitisation in genetically predisposed subjects and exposure to concentrations of more than 10µg/g a risk factor for acute attacks of asthma (85-86). Similarly, concentrations of greater than 8µg/g of *Fel d* I is indicative of a cat in the house and may be a risk level for symptoms (87-88). The concentrations of these and other allergens vary enormously between houses and environmental factors governing their distribution and concentrations may be important in determining the expression of allergic diseases.

House dust mite allergens occur throughout the world, but climate, season, outdoor temperature and humidity can affect both species of mite found and concentrations of indoor mite allergens. In general, higher levels of mite allergen are found in houses in hot, humid climates (89-90) and lower levels in colder winter months (91), dry arid areas (92) and at high altitudes (93-94).

There are also large variations in mite allergen concentrations in different sites within the home and between homes in the same geographical location. Local indoor environmental factors such as indoor temperature and humidity and condition of the house may also be important in determining allergen concentrations (95). Whilst most houses have indoor humidities greater than the minimum absolute humidity necessary to support mite growth (96), damp housing conditions and increased indoor humidity are associated with increased house dust mite populations (97-98) and allergen levels (99-100).

A damp indoor environment is also more likely to support mould growth and subjects living in houses with evidence of mould are more at risk of developing allergic symptoms (101). Subjects living in damp homes are therefore likely to be exposed to higher concentrations of allergens important in the development of asthma. One would therefore expect to see a higher prevalence of asthma in subjects living in such housing conditions, which furthermore may also adversely affect asthma severity. Poor housing conditions are known to adversely affect health, but their influence on the development and severity of asthma are poorly understood. The aim of this thesis was to investigate the relationship between asthma and damp housing in more detail.

Chapter 1

Section II

Housing characteristics and health

1.4 Historical background

An association between poor housing and ill health has long been recognised. To the Victorians, the causal links between poor housing conditions such as overcrowding, inadequate fresh water supply and sewage disposal and ill health were indisputable. Infectious diseases such as dysentery, cholera and tuberculosis were all commoner in families from poorer homes. Various Acts of Parliament were introduced to address these problems culminating in the Housing of the Working Classes Act (1898). This legislation often resulted in slum clearances which led to an improvement in public health.

In 1912, Dr Williamson, Minister of Health for Edinburgh, described the removal of families from old tenement housing to new cottages and reported a significant fall in mortality rates (102). Other re-housing projects did not always meet with similar success. McGonigle in 1933 (103) reported higher morbidity and mortality in rehoused individuals. This was thought to be due to the financial hardship experienced by the families moved to the new housing estates and indicates the complex relationship between housing quality, other socio-economic variables and health.

After the first world war, houses in Britain were in short supply and a huge building programme began to provide "homes fit for heroes". Following the second world war housing again became a major issue. Further clearance of inner city slums was required partly through damage from bombing and partly due to general disrepair. These slum clearances were expected to eradicate poor sanitation and most other precursors of ill-health. This reasoning meant that as the housing programme continued, health issues would be likely to prove of less concern. Indeed in 1951 health and housing policies were finally divorced when the housing responsibilities of the Ministry of Health were transferred to the newly created Ministry of Housing and Local Government (104).

The emphasis on the country's housing programme over subsequent years shifted from quality to quantity. This trend reached its peak in the 1960's when large rapid-build housing projects were utilised, such as concrete high-rise flats. These homes were often of poor design and not structurally sound. They were hard to heat, and prone to damp and condensation. Although occupants of these homes complained about their living conditions, authorities more often than not blamed the victim for causing the adverse living conditions.

At the time there was only limited scientific evidence to support the widespread belief of an interdependent relationship between housing quality and health (105). Although poor housing was perceived to be associated with poor health, both were associated with low income, poor nutrition, overcrowding and lack of education. This made it difficult to measure the relationship between housing and health. This relationship was often strongest for communicable diseases, particularly respiratory diseases such as tuberculosis, influenza and pneumonia. These were predominantly airborne infections and were closely associated with indices of overcrowding.

Little was known of the effects of housing deficiencies such as damp or inadequate heating on the development of chronic diseases. Duvall in 1978 (106) investigated the effects of housing conditions on women's health and found the most important factor was minor structural housing deficiencies such as broken windows, minor cracks in walls and poor decor. Insufficient heating during the winter months was also found to adversely effect physical health, as women living in such homes had more days sick in bed. In a more recent study, self-reported damp housing was associated with adult ill health and chronic disease (107). This observation was also seen in non-manual social classes living in owner occupied housing. Several of the associations with chronic illnesses persisted after controlling for confounding socio-economic variables. They concluded that poor housing conditions may adversely influence health independently of socio-economic status.

Dampness in the home may affect mental as well as physical health (108). The analysis in this study used subjective measures of housing conditions. It could be argued that respondents with poorer mental health were more likely to perceive and report their housing conditions as poor, although the study did not confirm poor mental health to be associated with all adverse housing conditions.

Much of the public housing in Britain today is in a state of disrepair (109-110) and is now recognised to have the potential to adversely effect the occupant's health. This poor quality of the household environment may be a contributor to ill health and housing is again becoming a public health issue. A recent survey of directors of public health in Britain confirmed that one third perceived housing as a major health problem, with positive responses more likely from inner city districts (111). Almost half of the departments surveyed allocated formal time commitment to housing issues, although in the majority of cases the main function of this was allocation of medical priority for public sector rehousing. The authors concluded that more time was required to be allocated to establish a health promoting housing policy.

The recent report "The Nation's Health, A strategy for the 1990's" (112) stated that there was "clear evidence linking bad housing to poor health and improvement of our housing stock is a priority in itself that would be likely to result in improvements in the quality of life for many of the most deprived in the community." It acknowledged that improving the housing stock was a priority for improving health but no specific recommendations were proposed as to how this might be achieved. With reference to respiratory disease and in particular asthma, it would be useful first to identify which aspects of adverse housing were important in contributing to the development of the disease and its severity so that programmes could be implemented to correct them. The influence of specific adverse housing conditions on respiratory symptoms and in particular asthma will therefore be discussed in detail below.

1.5 Indoor temperature, humidity and health

Most people believe living in a cold, damp home is bad for their health, but scientific evidence to support this claim is weak. An excess of deaths in Britain during the winter months has been documented. Although the World Health Organisation (WHO) recommended a minimal indoor temperature of 18°C, some household surveys in Britain found winter indoor temperatures as low as 6°C, with 75% of the elderly living in homes with temperatures below the WHO recommendation (113).

The recorded monthly death rates for England and Wales during 1962-67 identified a relationship between environmental temperature and the death rate (114). The time between the onset of a cold spell and an increase in mortality was 1-2 days for myocardial infarction, 3-4 days for cerebro-vascular accidents and 7 days for pneumonia and bronchitis. In a more recent study, it was calculated that for every 1°C drop in average winter temperature, there were approximately 8000 excess deaths in Britain (115). Such large excess winter mortality rates are not seen in countries which have more severe harsh winters, probably reflecting the better insulation and heating in their houses (116).

In numerical terms, hypothermia accounts for only a small proportion of these excess winter deaths, which occur predominantly in the elderly. Death from coronary and cerebro-vascular disease both increase during winter months. Prospective studies have shown that plasma fibrinogen levels predict the development of

cardiovascular disease (117). Seasonal changes in fibrinogen levels occur in subjects aged 75 years and over (118), when plasma fibrinogen levels were greater in the coldest months and inversely related to core body temperature and environmental temperature. These changes may explain the increased risk of cardiovascular disease in winter.

The elderly, children, handicapped, unemployed and chronic sick are all particularly vulnerable to low indoor temperatures as these groups tend to spend the majority of their time at home. They are also more likely to have lower incomes and be less able to adequately heat their homes. The habit of heating only part of the dwelling is common and may predispose to condensation and damp within the home, resulting in further adverse health effects.

Higher indoor relative humidities favour the growth of moulds and bacteria, favour house dust mite growth and increase survival of air borne pathogens. Respiratory illness and the common cold are associated with high relative humidity and damp housing conditions, but the findings of such studies and in particular those relating to upper respiratory tract infections, have been inconsistent (119-122).

1.6 Damp housing in Britain: extent of the problem

A Scottish house condition survey was recently undertaken to give a comprehensive, national picture of Scotland's housing stock (123). It estimated there were just over two million occupied dwellings in Scotland and a full physical survey was carried out on a random, representative sample of 15,272. Twenty-eight percent of surveyed dwellings (an estimated total of 584,000 dwellings) had evidence of damp, condensation or mould growth either singly or in combination. Approximately 5% dwellings surveyed (an estimated total of 95,000 dwellings) were below tolerable standard as defined by the Housing (Scotland) Act 1969 and set out in part IV of the Housing (Scotland) Act 1989. This states that a dwelling can fail the Tolerable Standard on any one of nine criteria covering dwelling condition and amenities. Almost three-quarters of dwellings failed on one item, most commonly because the dwelling was not "substantially free from rising or penetrating damp."

A comparison of damp across the regions revealed Strathclyde with the highest proportion of damp dwellings. A smaller study had previously found 47% surveyed dwellings in Glasgow had signs of disrepair associated with damp (97). These

conditions occurred more often in unmodernised flats, old tenement buildings and 1960's council housing stock which are often cold as well as damp. Such homes are often occupied by people on low incomes such as the unemployed, chronically sick and elderly, who are the least able to afford to heat their dwellings adequately. Similar surveys in England and Wales estimated that between 25 - 33% of homes are affected by damp (110,124).

1.7 Causes of damp in dwellings

Dampness in a dwelling may be classified by either the source or the route by which the water enters. A distinction is usually made between water which enters a building material as a liquid, as occurs in rising or penetrating damp or faulty plumbing, and that which is condensed from the atmosphere - condensation.

1.7.1 Rising and penetrating damp

Rising damp results from the capillary flow of water from the ground and is usually prevented by the installation of a damp proof course in the foundations of the dwelling. It's absence or a breach of an existing damp course will result in rising damp, often extending about 50 cm above skirting board height. Direct rain penetration through the walls of the dwelling may also occur, either due to the high porosity of the brick or faulty brickwork. Damp due to this cause is usually found on external south or south-westerly facing walls in British dwellings. Rising and penetrating damp are more frequent in older dwellings and were found in 13% homes surveyed in Scotland in 1991(123).

1.7.2 Condensation

Until the 1960's, condensation was rarely found in dwellings, but with recent changes in building design and living conditions, this source of damp has become a major problem. In one survey, condensation accounted for approximately two-thirds of complaints regarding damp received by local housing authorities in England and Wales (109). Condensation was found in 19% of occupied dwellings in Scotland (123). The high prevalence of condensation in British dwellings merits further discussion.

Air always contains some moisture, which contributes to the total atmospheric pressure. This contribution is called the vapour pressure of the water. The greater the temperature of the air the more water vapour it can hold. When air holds the maximum amount of water vapour possible, it is said to be fully saturated. The relative humidity (RH) is the amount of water vapour in air expressed as a percentage of the water vapour when saturated. Saturated air therefore has a RH of 100%. Air containing only half of the moisture it could contain at that temperature would have an RH of 50%. If air is cooled, the amount of water vapour it can hold is reduced and it's RH increases. The air temperature where the RH reaches 100% is referred to as the dew point, as any further reduction in temperature will result in excess water vapour condensing as dew.

The dew point temperature is therefore important whenever condensation is concerned. Dew point temperature increases as the moisture content of the air increases. Accordingly, the more moisture within a dwelling, the more likely that the dew point temperature will increase beyond that of an indoor wall surface temperature, resulting in condensation. When the dew point occurs within walls, floors or construction layers, it is referred to as interstitial condensation.

Condensation does not arise because of high outdoor humidities. As long as indoor temperatures are even slightly greater than those outdoors, air entering from outside can never reach saturation. Thus if all the moisture produced within a dwelling escaped, condensation would never occur.

Many of the changes in building design over the past thirty years have been driven by energy conservation, so that dwellings have become more air-tight with reduced ventilation. This has had the benefit of reducing expenditure on fuel, but has resulted in dwellings more prone to condensation. Another phenomenon causing condensation is cold-bridging where local cold areas are generated on otherwise warm walls by the proximity of highly conductive building materials such as concrete lintels, which were often used in the construction of the tower blocks in the 1960's. These cold areas can be as much as 5°C lower than the surrounding wall temperatures, predisposing to the formation of condensation.

The major sources of moisture production within dwellings are shown in table 1. Moisture production is proportional to the number of occupants. High moisture generating activities include cooking and the drying of clothes indoors using either an unvented tumble drier or clothes-horse. The use of unvented gas or paraffin heaters is an uncommon, but important source of moisture production. Heating is generally not sufficient to prevent condensation, but may delay it's occurrence as the warmed air takes longer to cool at any cold surface, giving any existing ventilation

more time to replace the wet air with drier air from outdoors. Condensation could also be prevented by raising the temperature of the cold surfaces. This however is often limited by the available heating system and its running costs. The common practice of using heating intermittently during the evenings and mornings is unlikely to significantly reduce the risk of condensation.

The presence of damp and condensation is therefore common in British dwellings today. The evidence that these housing characteristics influence respiratory health is reviewed in the next section.

Table 1. Typical moisture generation rates for household activities.

(Reproduced from British Standards 5250: 1989 Appendix B.)

Household Activity		Moisture Generation Rate
People:	Asleep	40 g/hr per person
	Active	55 g/hr per person
Cooking:	Electricity	2000 g/day
	Gas	3000 g/day
Dish washing		400 g/day
Bathing / washing		200 g/person per day
Washing clothes		500 g/day
Drying clothes indoors		1500 g/person per day

Chapter 1

Section III

Damp housing and respiratory illness

1.8 Respiratory symptoms and damp in the home

Sir John Floyer (1649 - 1734), an English physician who suffered from asthma, published *A treatise of the Asthma* in 1698 (125). This work and its contribution to our understanding of the disease has been summarised by Sakula (126). Floyer observed over 250 years ago that “damp houses and fenny countries” were bad for asthma. He was one of the first to document respiratory symptoms induced by damp when in 1726 he described an acute attack of asthma on visiting a damp wine cellar (127).

Over the past thirty years, there have been many studies from countries throughout the world reporting a higher prevalence of respiratory symptoms including wheeze in adults and children living in damp dwellings. These studies are summarised in table 2.

Environmental factors such as adverse housing conditions were implicated in the development of respiratory disease in children by Holland *et al* in 1969 (128). The ventilatory function (peak expiratory flow rates) of 10,971 schoolchildren was found to be influenced by environmental factors such as area of residence, social class and family size. Tentative conclusions were that the differences between children living in different areas were in part due to their housing conditions.

A later study of respiratory health of children in South Wales (129) claimed that children living in new, centrally heated council homes had more respiratory symptoms and poorer lung function than those in traditional valley housing. This was an unexpected finding for which there are several possible explanations. Fewer mothers in the traditional housing areas were cigarette smokers and it is likely their children were exposed to less passive smoking. There was also an excess of reported upper respiratory infections in children from the new council houses which may have accounted for their impaired lung function. Finally, the new centrally heated council homes provided better growth conditions for the house dust mite and may have increased the prevalence of allergic diseases such as asthma.

Twenty years later, respiratory illnesses are still being found to be associated with socio-economic deprivation. Spencer *et al* found that residence in an area of social and maternal deprivation increased the risk of admission to hospital of one year old children with suspected bronchiolitis (130). This particular association was not influenced by either parental smoking or disease severity and was likely to be due to some other adverse condition in the home environment.

Table 2. Risk of respiratory symptoms in subjects living in damp or mouldy dwellings

Study	Country	n	Age (yrs)	Housing characteristics	Risk of respiratory symptoms
Strachan 1986	U.K.	165	7	Mould	↑O.R. 2.7 - 3.9
Martin <i>et al</i> 1987	U.K.	358	<16	Damp/mould	↑ O.R. 3.7
Strachan 1988	U.K.	873	7	Damp/mould	↑O.R. 3.0
Andrae <i>et al</i> 1988	Sweden	4990	1 - 16	Damp	↑ RR 1.9 ↑↑if also ETS
Waegemaekers 1989	Holland	519	>16	Damp	↑RR2.8 (wheeze) ↑RR6.1 (Allergy)
Brunekreef <i>et al</i> 1989	U.S.A.	4625	8 - 12	Damp/mould	↑RR 1.3 - 2.2
Platt <i>et al</i> 1989	U.K.	1169	<16	Mould	↑ O.R. 1.2 - 1.9
Hyndman <i>et al</i> 1990	U.K.	60	>16	Mould	↑RR 2.5 - 3.0
Dales <i>et al</i> 1991	Canada	13495	<16	Damp	↑RR 1.3 - 1.9
Dales <i>et al</i> 1991	Canada	14799	>16	Damp	↑RR 1.3 - 1.9
Brunekreef <i>et al</i> 1992	Holland	3344	6 - 12	Damp/mould	↑RR 1.5 - 1.7
Jaakkola <i>et al</i> 1993	Finland	2568	1 - 6	Damp/mould	↑RR 2.5 - 6.9
Spengler <i>et al</i> 1994	U.S.A	600	9 - 11	Damp	↑RR 1.2 - 1.6 (dose-response)
Cuijpers <i>et al</i> 1995	Holland	470	6 - 12	Damp	↑RR 3.0 (cough in boys only)
Timonen <i>et al</i> 1995	Sweden	2564	7 - 12	Damp	↑RR 2.5 (wheeze) Dose-response
Austin <i>et al</i> 1997	U.K.	1537	12-14	Damp/mould	↑RR 1.6 (cough)

O.R. = odds ratio. R.R. = Relative risk. ETS = Environmental tobacco smoke.

The relationship between the home environment and respiratory morbidity was further investigated by Strachan in Scotland (131). Twelve features of the home environment and the prevalence of respiratory symptoms were studied in 165 children age 7 years. Three factors independently contributed to the risk of wheeze: a family history of wheeze, an open bedroom window and mouldy housing. The latter was also a risk factor for nocturnal cough. However, none of the environmental

conditions were associated with general practise consultations for wheezing or respiratory illness.

In a larger study, Strachan *et al* surveyed 873 seven year old children and found an association between parental reporting of damp and mould in the home and respiratory symptoms in their children (132). Again there was no correlation between reported symptoms and general practitioner consultations for respiratory complaints. Furthermore, there was no correlation between reported symptoms, exercise induced bronchoconstriction and self-reported damp in the home. The authors concluded that reporting bias by the parents was playing a large part in the observed association.

The same study also measured bedroom temperature and relative humidity in a stratified sample of 317 children (133). There was no correlation between these measurements and reporting of respiratory symptoms including wheeze, measures of airflow obstruction or exercise induced bronchoconstriction. These results contradicted the findings of Melia *et al* (120) who observed a significant positive association between the prevalence of respiratory conditions in five and six year old children and indoor relative humidity. Strachan argued that reported dampness was a poor predictor of measured humidity and suggested that damp due to condensation would also be highly dependent on the temperature of indoor walls and surfaces as well as air humidity. Measuring temperature and relative humidity at the wall surface may therefore be a more appropriate measure of damp in the home.

Risk factors for childhood respiratory disease were studied by Schenker (134) in a survey of 4071 children aged 5 to 14 years in the USA. Male sex, lower socio-economic status and a positive parental history of allergy or respiratory disease were all identified as independent risk factors for most respiratory symptoms and illnesses. Passive smoking was associated with a history of chest illness before two years of age. Although it had been speculated that nitrogen oxide emitted from the use of gas cooking stoves may induce respiratory symptoms, this study found the use of gas stoves was inversely related to socio-economic status and not an independent risk factor for respiratory symptoms or illnesses. Other physical aspects of the home environment such as the presence of damp or mould were not included in the study.

Dales *et al* conducted a large questionnaire-based study on the health effects of the indoor environment of 13,495 school children aged 5 to 8 years (135) and 14,799

parents (136) in thirty Canadian communities. Thirty-eight percent of homes had evidence of damp or mould. The prevalence of lower respiratory symptoms was increased in those subjects reporting damp in the home with adjusted odds ratios ranging from 1.3 to 1.9. A similar study examining the relationship between measures of home dampness and respiratory illness was conducted in a cohort of 4,625 eight to twelve year old children in six U.S. cities (137). One or more of the dampness indicators were present in over half of the homes and a strong, consistent association was observed between respiratory symptoms and the presence of damp and mould. Adjusted odds ratios for moulds ranged from 1.3 to 2.1 and for dampness from 1.2 to 2.2.

Spengler *et al* conducted a study of respiratory symptoms and housing characteristics in 600 children aged 9 to 11 years, living in twenty-four communities throughout the USA and Canada (138). Approximately half of the households reported damp conditions in the home. Home dampness had a strong association with bronchitic symptoms (odds ratio 1.6) and a weaker association with asthmatic symptoms (odds ratio 1.2) across all cities. Odds ratios were higher when two or more rooms had mould versus a single room, suggesting a dose-response relationship. Better educated parents with higher incomes and having asthma were more likely to report damp conditions. Even allowing for this, there was no evidence of a socio-economic trend for the association of respiratory symptoms and home dampness.

A pilot study in Holland (139) investigated the association between living in damp homes and the prevalence of respiratory symptoms in a population of 519 occupants of 185 homes. Indoor fungal spore counts were measured in a sample of 36 homes. Forty-two percent of the homes were classified as damp using a checklist for signs of damp. Respiratory symptoms were more prevalent in both adults and children living in damp homes, adjusted odds ratios varying between 2.8 for wheeze to 6.1 for allergy. Fungal spore concentrations in living rooms were higher in damp homes and correlated with subjective dampness perception. This study contained a potential for respondent bias, as the study was promoted by the occupant's concern. However the association between symptoms and objective measures of fungal spore counts in a sub-sample of the home mitigates against this being of major concern.

Brunekreef administered questionnaires to 3344 parents of 6 to 12 year old children in Holland (140). Twenty-five percent of homes were reported to have visible damp

stains or mould. Cough and phlegm in men and women were strongly associated with living in a damp home, even after controlling for the effect of smoking. Weaker associations were found for wheeze and asthma. Several years later, Cuijpers *et al* (141) also studied the effects of exposure to various factors from the indoor environment on the respiratory health of 470 Dutch school children aged 6 to 12 years. They found the presence of damp stains in the home was associated with chronic cough in boys (O.R. 3.0), but not in girls. Dampness in the home was not associated with any other respiratory symptoms, including wheeze or attacks of dyspnoea.

In a recent study of the prevalence of respiratory symptoms in children living in the Scottish highlands (142), 1537 questionnaires were completed by the parents of 12 to 14 year old children. The symptom of cough was associated with damp (relative risk 1.6) and mould (relative risk 1.8) in the home. Neither the method of heating or any other environmental factor appeared to influence the prevalence of current wheeze.

Similar cross-sectional studies have been conducted in Scandinavian countries, where the prevalence of asthma is generally lower than in Britain. In a Swedish study, the parents of 4990 children aged 6 months to 16 years completed a validated questionnaire on environmental factors and symptoms of bronchial hyperreactivity and allergic disease (51). A representative sample of 34 houses were visited by health inspectors to evaluate structural damage due to dampness. Children living in damp houses were more likely to have bronchial hyperreactivity (relative risk 1.9) and asthma (relative risk 1.9). This association was strongest in children with a family history of allergy and was exacerbated by parental smoking (relative risk 2.8). Although only fourteen houses were examined in detail, damage due to damp was largely equatable to indoor visible mould sources. House dust mites were found in low numbers in four houses and were felt unlikely to be contributing to symptoms.

A study of 2568 children aged 1 to 6 years in the Helsinki area of Finland found associations between respiratory symptoms including cough and wheeze and signs of damp in the dwelling (143). The presence of mould odour during the past year and water damage over one year ago had the strongest association with respiratory symptoms (Odds Ratios ranging from 2.5 to 6.9). The occurrence of respiratory symptoms was related to the frequency of days mould odour was reported indicating a dose-response relationship. There was no association between children with current asthma diagnosed by a doctor and any of the dampness indicators, (odds ratio 1.1). The prevalence of asthma was however low, at 2% of the study population.

In the latest Scandinavian study (144), the parents of 2564 school children aged between 7 to 12 years completed a questionnaire regarding the presence of chronic respiratory symptoms in their children and the presence of environmental risk factors such as moisture stains or mould in the home. Again, the parents of children with asthmatic symptoms (wheezing and shortness of breath) reported damp more often than parents of asymptomatic children, odds ratio 2.5.

All the studies described above report an association between measures of self-reported damp in the home and an increased risk of wheeze in adults and children, but most are open to the same criticism that the results may be influenced by reporting and recall bias. It is possible subjects with respiratory symptoms are more likely to be aware of adverse housing conditions and attribute their symptoms to living in such conditions. They would then be more likely to answer positively to questions regarding their health. Some investigators have attempted to overcome this problem by obtaining independent, objective measures of damp in the home.

Martin *et al* interviewed 358 tenants in Edinburgh, Scotland and environmental health officers independently surveyed 300 of these households (145). Twenty-four percent of the dwellings were damp and 17% had mould growth. There was a significant association between living in damp conditions, especially mouldy housing, with respiratory ill health in children, but not in adults. The association remained after controlling for passive cigarette smoking and overcrowding. The study design minimised respondent bias by having an independent survey of damp. Differential over-reporting of symptoms by those in damp housing was not substantiated by the study findings as respondents who reported damp in the home were no more likely to report symptoms in themselves or their children than those from dry homes.

In a larger study the same group surveyed 597 households living in public housing in Edinburgh, Glasgow and London (146). The study was of similar design with independent surveyors assessing each home for damp and mould growth. In addition, air spore concentrations were also measured in the Scottish homes. Sixty-nine percent of dwellings were affected by damp or mould. An increased prevalence of non-specific symptoms occurred in adults living in such conditions. Children were more likely to have respiratory symptoms such as wheeze, sore throat or runny nose if they lived in damp homes. The data supported a dose-response relationship since a significant tendency for increasing severity of dampness and mould growth in the dwelling was associated with a greater prevalence of non-specific symptoms in children and adults.

Hyndman also used objective measures of dampness when investigating a possible association between damp public sector housing in London and the health of 60 British Bengali households (147). There was a high prevalence of damp, which was significantly worse in non-centrally heated homes. Respiratory symptoms suggestive of asthma were associated with lower indoor temperatures (relative risk 1.5) and with reported and observed mould growth (relative risk 2.5 to 3.0). The combined effect of living in a cold damp environment produced generally poorer health. The prevalence of such living conditions was much higher in non-centrally heated homes, implying that household characteristics were less important than the provision of adequate heating.

1.9 Damp housing and lung function

Few studies of the association between damp housing and respiratory ill health have included objective measures of respiratory disease. Most found no association between damp in the home and measures of lung function such as forced expiratory volume in one second (FEV₁) (148) and peak expiratory flow (PEF) (147). However, in a study of 470 Dutch school children (141), the PEF was approximately 3% lower in boys living in homes with evidence of damp ($p < 0.05$), but no difference was observed in the lung function measurement in girls. A study of 4625 American school children aged 8 to 12 years also reported a slight impairment in lung function in the form of a reduction in mid expiratory flow (FEF_{25-75%}) in children living in homes with visible mould growth. There was no difference in the level of FEV₁ or FVC with home dampness (137).

Strachan correlated the presence of exercise induced bronchial lability in 7 year old school children with parental reporting of damp in the home (132). No significant differences were observed in the degree of bronchospasm measured among children from homes with and without mould. He concluded that the relationship between mould and wheeze was unrelated to airway reactivity. However, significant bronchoconstriction after exercise only occurred in a small proportion (2.3%) of the children. In a follow up study (149), airborne mould spore counts were measured in a subgroup of 88 children's homes. Twenty-six children had significant exercise induced bronchoconstriction. A small non-significant increase in the total airborne mould counts was observed in the homes of these children.

1.10 Damp housing and asthma

Although individually each of the studies summarised in Table 2 has its limitations, they consistently point to an association between respiratory ill-health and living in damp or mouldy living conditions. None have been able to identify an association with any specific disease entity such as asthma, as there is no currently validated questionnaire with which to diagnose asthma in epidemiological surveys. Few conclusions can therefore be drawn from these cross-sectional, population based studies with regards any possible associations between asthma and damp housing. A case-control study design would overcome some of these methodological difficulties. There have been several such studies, the results of which are summarised in table 3.

Table 3. Damp and mouldy housing and the risk of asthma

Study	Country	n	Age (yrs)	Housing characteristic	Risk of asthma
Leen <i>et al</i> 1994	Ireland	134 cases 118 controls	<16	Temperature and RH	→ OR
Fagbule <i>et al</i> 1994	Nigeria	140 cases 140 controls	<16	Damp/mould in bedroom	↑OR 11.2
Mohamed <i>et al</i> 1995	Kenya	77 cases 77 controls	9 - 11	Damp	↑OR 2.1 - 4.9
Lindfors <i>et al</i> 1995	Sweden	193 cases 318 controls	1 - 4	Damp, pets & ETS	↑OR 1.9 - 8.0
Verhoeff <i>et al</i> 1995	Holland	259 cases 257 controls	6 - 12	Damp/mould	↑OR 1.5 - 2.9

OR = Odds Ratio. ETS = Environmental Tobacco Smoke.

In the Netherlands in 1990 two hundred and fifty-nine children with chronic respiratory symptoms and 257 control children were studied (100). A strength of this study was that all homes were inspected for signs of damp and it did not rely solely on measures of self-reported damp. The cases were more likely to live in a

home where damp had been observed or reported. In both cases and controls, home dampness was associated with increased sensitisation to dust mites and moulds. The odds ratios were increased by restricting the analysis to those controls with normal IgE levels and cases with elevated total IgE (who were likely to be asthmatic). This study suggests damp housing may increase the prevalence of childhood asthma by increasing sensitisation to house mites and moulds.

Leen *et al* in Ireland administered a questionnaire on the home environment to the parents of 134 children with asthma and 118 controls (150). A detailed study of the child's' home environment was performed in only a sample of 10 cases and 10 controls. Temperature, humidity and dust mite allergen levels were similar in both groups. Mite allergen levels were generally high and the authors concluded that all the children were exposed to a high allergen load. However, the sample of homes studied was small and may have under-estimated differences in exposure to mite allergen levels between the two groups of children.

Two other case control studies originating from the African continent reported an association between damp housing and asthma. Fagbule *et al* found that asthmatic Nigerian children were more likely to sleep in a damp, mouldy bedroom than age and sex matched controls, odds ratio 11.2 (151). Mohamed *et al* reported that asthmatic Kenyan schoolchildren aged 9 to 11 years were more likely than age and sex matched controls to live in homes with evidence of damage caused by dampness, odds ratio 2.1 and in particular to have evidence of damp in their sleeping area, odds ratio 4.9 (68). This study also found that a higher level of indoor air pollution and a higher supplemental salt intake were also associated with asthma.

The most recent case-control study was performed in Sweden (152), where indoor environmental risk factors were compared in 193 asthmatic children and 318 controls aged one to four years. Measures of self-reported damp such as window condensation or damage secondary to damp were associated with asthma, especially in children sensitised to cats or dogs, odds ratio 1.9. Other significant factors included exposure to environmental tobacco smoke and allergy to household pets such as cats and dogs. There was a significantly increased risk of asthma with increasing number of risk factors such that if all three risk factors coexisted, the odds ratio increased to 8.0.

Home environmental factors including damp housing conditions may therefore be important in the development of allergic disease such as asthma in both developing

and industrialised countries. However, the evidence for this is largely derived from observational epidemiological studies. Such studies are vulnerable to bias and confounding. Bias commonly arises due to faults in study design and may lead to a misrepresentation of the relation between exposure and disease. Confounding may occur if the exposure is associated with other risk factors for the disease. These will be discussed further in the next section.

Chapter 1

Section IV

The influence of bias and confounding on case-control study design

1.11 Bias and confounding in case-control studies

The case-control study design has become increasingly popular in epidemiological research in recent years. It's principal objective is often to provide a valid and reasonably precise estimate of the strength of a hypothesised cause-effect relationship, for example between damp housing and asthma. An important limitation of such studies is their susceptibility to bias, which creates non-comparability between cases and controls. Sackett catalogued 35 potential biases in sampling and measurement in case-control studies (153). These fall broadly into two categories.

First, selection bias may arise if the individuals for whom data is available are not representative of the study population. This is important in asthma epidemiology, as the study population selected will depend very much on the definition of asthma adopted for that study. Failure to choose a representative sample (sample bias) or failure to obtain information on all members of a sample (non-response bias) may also impair the validity of the findings.

Second, information bias may arise from shortcomings in collecting, recording or analysis of the data. Observer bias may arise in collecting the information, and recall bias in those under study, for example if cases give more thought to questions than controls. This is of particular concern in studying the relationship between damp housing and asthma. Asthmatic subjects may be more aware of adverse housing conditions such as damp and mould and associate these with their poor health. As a result they are likely to respond differently to questionnaires regarding housing and health than control subjects. This has been cited as a particular concern by Strachan (132), but can largely be overcome by independently assessing housing conditions. Where possible the direction and magnitude of any possible bias should be appraised so that appropriate allowances can be made.

In addition to bias, the possibility of confounding must also be taken into account when analysing epidemiological studies (154-155). Although the evidence suggests that exposure to damp housing is associated with respiratory symptoms and asthma, this association may exist because damp housing is associated with other risk factors for asthma. For example, living in damp, mouldy housing conditions is frequently associated with poor socio-economic status. Such subjects are frequently of lower social class, more likely to be unemployed and to have lower household

incomes. Chronic sickness such as asthma may also limit employment opportunities, resulting in lower incomes and these individuals may gravitate to poorer housing. The problems of confounding can often be overcome, either by matching of cases and controls at the stage of study design or during statistical analysis. This is only possible if confounding factors are recognised and dealt with. When investigating the relationship between damp housing and asthma, any association between asthma and lower socio-economic status may act as a potential confounding variable and this merits further discussion.

1.12 Income, unemployment and health

Socio-economic inequalities in health stem, at least in part, from inequalities in income. The relationship between income and health is not linear, as a given reduction in an individual's income results in a larger deterioration in health, for those individuals on lower incomes (156). The Registrar General's social classes are groupings of occupations with a wide range of incomes (as well as some unemployed) in each class. The income differences within each class tend to be as large as those between classes. To look at this issue, Wilkinson matched mortality and income for 64 occupations using the 1971 and 1981 census data (157). Unemployment and low wages accounted for 21% of the variance in mortality. The data supported the hypothesis that the association between relative poverty and mortality is at least partly causal and is responsive to changes in income. The relationship between unemployment and health is likely to be a reflection of the low incomes of those living on unemployment benefit.

Involuntary unemployment may result in deterioration in the mental and physical health of those concerned. Cook *et al* questioned 7735 men aged between 40 - 59 years on employment and health (158). 408 (5.3%) of the men were unemployed and a distinction was made between those unemployed due to ill health or for other reasons. The unemployed men had far more self-reported physical illnesses than the employed, whether or not they regarded themselves as unemployed due to ill health. The extent to which unemployment carries a risk to health is difficult to assess. Those who are disadvantaged by unemployment also tend to have low incomes when in work, live in poorer housing conditions and have comparatively little education. Failure to consider these factors may result in wrongly attributing poor health to unemployment.

Chronic illness such as asthma may limit employment choices and opportunities. Ross *et al* studied the social consequences of wheeze in childhood over a 25 year period (159). They found that childhood wheeze did not adversely effect educational attainment, employment, housing or social class in the population studied. However, the majority of children had only mild to moderate asthma severity and it is likely that those with more severe asthma would have a higher rate of unemployment, lower incomes and hence poorer housing conditions.

Asthma severity correlates statistically with work disability in adults (160). Over a two year period, 42 adults aged 18 - 55 years were questioned with regards to job duties, reduction in pay, and change in job or employment status attributed to their asthma. Their asthma severity was scored on frequency of asthma symptoms and medication and correlated well with lung function measurements. The 5 year cumulative incidence of work disability was 19% for changes in duties, 17% for reduction in pay, 20% for change in job or work status and 36% for any of these measures. The severity of asthma score was correlated with each measure of disability implying that those with more severe asthma were more likely to have a higher degree of disability which could significantly alter their employment opportunities.

1.13 Asthma and social class

Inequalities in health were highlighted by the publication of the Black report in 1980 (161). The central findings were large differentials in mortality and morbidity favouring higher social classes. Despite highlighting these differences over a decade ago, these social class differences in mortality are widening (162). It is likely that these class differences in health are due to differences in lifestyle and living conditions rather than from differences in health care utilisation.

In general, respiratory disease is more prevalent in the lower social classes and is likely to be due to greater exposure to aetiological factors such as tobacco smoking and poor housing. The relationship between asthma and social class is less clear. Early studies suggested asthma was commoner in the higher social classes (163-165), although severe asthma was found to be more common in lower social groups (166-167). The latter effect has now also been confirmed in more recent studies.

In a study of disabled adults in England and Wales in 1990 (168), 338 reported asthma as a contributing cause of their disability. Overall, adults in social classes 4 and 5 were twice as likely to have disabling asthma compared to adults from social classes 1 and 2. This could be due to a higher prevalence of asthma in lower social classes, but this is not borne out by other studies. Alternatively, increased morbidity secondary to greater exposure to exacerbating factors is a more likely explanation. Inadequate treatment of asthma in lower social groups could also contribute to these findings. The same severity of asthma may be considered more disabling by adults from manual occupations, but the authors found a similar trend for women whose social class was classified according to their partners occupation, mitigating against this explanation.

Social class differentials were reported in a retrospective analysis of mortality from asthma in England and Wales between 1979 to 1987 (169). Death rates were higher in men in manual occupations. An age-dependant, geographical variation in asthma deaths was found, with a higher mortality in younger subjects (5 - 34 years) in the South of the country and significantly higher in older subjects (35 - 64 years) in the North. Such a pattern of regional variation is not compatible with exposure to a single environmental aetiological exposure.

Strachan *et al* recently identified a higher level of asthma morbidity in children from poorer families (170). In a nation-wide survey of 3209 families, 15% of children had wheezed in the past twelve months. Whilst the prevalence of wheeze did not differ by social class, there were marked trends in the prevalence of frequent and severe attacks and nocturnal symptoms were greater in children from lower social class families. They suggested that social factors may affect the severity of the condition rather than it's prevalence. It was postulated that this effect may reflect adverse environmental factors such as exposure to tobacco smoke or poor housing.

People in lower social classes may be less healthy because they are socially deprived and live in poor circumstances. Alternatively they may have moved down the social gradient because of ill health. Social class mortality differentials are often calculated from the Office of Population Censuses and Surveys (OPCS) published each decade. This census allocates social class on the basis of occupation. However, a significant number of people cannot be classified by these means. This group includes men who are permanently sick and unable to work and their mortality is high. Their exclusion affects statistics generated from this source, causing a health related

selection bias. For this reason it is difficult to estimate the effect of social class mobility from these censuses.

A study by Fox in 1985 (171) compared the mortality gradient by social class five to ten years before death with the gradient calculated using the social class at death and found them to be similar. He concluded that mortality gradients were not significantly influenced by health related mobility between social classes. However, this study considered men aged between 55 - 65 years and it is possible that there would be more social class mobility in younger age groups. Despite this, he strongly favoured environmental and behavioural factors to account for the social class differences in mortality rather than the possibility of downward social mobility.

Chapter 1

Section V

Damp housing and allergen exposure

1.14 Damp housing and asthma - biological plausibility

Biological plausibility is frequently alluded to in interpreting epidemiological associations. Whilst this is often helpful in explaining an association, it is often too easy to generate arguments to explain study findings. However, interpretation of study data should not seriously conflict with known facts of the natural history and biology of the disease under investigation. There are several plausible mechanisms which have been proposed to explain the possible relationship between asthma and damp housing.

1.15 House dust mite and asthma

There is a considerable body of evidence to suggest that allergic diseases such as asthma, rhinitis and eczema occur in genetically predisposed (atopic) individuals who are exposed to certain environmental allergens. House dust has been long recognised as an important source of allergen. In 1662 a Flemish physician recognised that inhalation of dust particles in air could lead to symptoms which would now be recognised as a typical allergic reaction. However, it was not until the 1960's that Voorhorst *et al* (81) reported the house dust mite to be the major source of house dust allergen.

House dust mites belong to the species Arachnida along with spiders and scorpions and at least 50 species are found in domestic house dust. In temperate climates the most common mites are *Dermatophagoides Pteronyssinus* and *D farinae* of the family Pyroglyphidae. They are approximately 400µm in length, have an average life cycle of three months and feed of desquamated human skin and the fungi that grow it. They have a relatively hard pervious outer layer and are sensitive to water loss. As their water content is in equilibrium with the atmosphere, the ambient humidity determines their water content. They thrive best at a relative humidity of 80% and temperature of 25°C. As the ambient humidity falls, the mites stop reproducing and become immobile and at levels below 50% they are likely to die.

Mites have a developed gastrointestinal tract and produce approximately 20 faecal pellets (average size 10-40µm) a day. These pellets readily become airborne during domestic activities, but quickly settle out when activity ceases. Although over 30 different mite allergens have been identified, only five or six predominate and are usually water soluble and associated with mite faecal particles. The major allergen

associated with *D pteryonnisimus* is *Der p I*, a single chain protein of 222 amino acid residues and a molecular weight of 25,000 Daltons, identified as a cysteine protease secreted from the mite gastrointestinal tract. *Der p I* is now used as the standard measure of exposure to house dust mite allergen in Britain.

1.15.1 House dust mite and climate

House dust mites are found in most inhabited areas of the world, especially in homes with carpets, inside mattresses and bedding, soft toys and soft furnishings. The number of mites in house dust is dependent on both the indoor and outdoor climate. There is a relationship between outdoor humidity and level of indoor *Der p I* measurements (90, 92, 172). In a population based study from Australia, six groups of children living in different areas of New South Wales were studied (173). There was a significant relationship between mean outdoor humidity and mean indoor *Der p I* measurements, with the lowest levels observed in a dry, hot inland area and highest levels in a semi-tropical area. They observed a dose-response relationship between exposure to *Der p I* and the risk of asthma. After adjusting for sensitisation to other allergens, the risk of asthma was doubled with every doubling of *Der p I* level, confirming the importance of exposure to this allergen in the development of asthma.

Several studies have identified a seasonal variation in house dust mite numbers, with low levels in winter. These have tended to be in countries where outdoor relative humidity falls to below 50% for prolonged periods, for example in USA (91, 174, 175), Canada (172) and Sweden (176). Seasonal variation in countries with warmer, damper climates are less easily demonstrated. In Spain, a seasonal variation in *Der p I* levels was detected in a part of the country with a humid climate, but not in an area with a sub-humid climate (90). In Britain, Karla *et al* found no seasonal variation in mite allergen levels in Manchester over a one year period (177). It is unlikely that indoor relative humidity in countries such as Britain falls low enough to influence mite growth.

Modern energy efficient homes with good insulation and reduced air exchange help to keep indoor temperature and relative humidity constant, even in cold, dry climates. Such changes in house design create a more favourable growing environment for the house dust mite and have been proposed as an explanation for the increase in HDM allergy, where previously this was rare (176). Mite allergen

levels in Swedish homes were associated with the difference in absolute humidity between indoor and outdoor air as well as with low air-exchange rates of the home, particularly the bedroom (178). The authors concluded that in regions with a cold winter climate, the air exchange rate of the home was important in determining levels of infestation with house dust mites. Whether this is also true for countries with a warmer winter climate such as Britain is unclear.

1.15.2 House dust mite and housing characteristics

Although HDM levels are related to climate and possibly to changes with the seasons, there is often a greater variation in HDM numbers between different houses in the same season, suggesting certain housing characteristics predispose to higher mite numbers. In 1952, even before the house dust mite was identified as an important source of allergen, it was found that 38 out of 49 patients with a primary sensitivity to dust lived within 160m of waterways (tributaries of the Thames) compared with 17 out of 49 controls (179). Since then several studies have identified certain housing characteristics associated with higher levels of house dust mites but the most important overall factor determining mite growth would appear to be indoor relative humidity.

Korsgaard found greater numbers of HDM in the homes of 25 newly diagnosed HDM sensitive asthmatics than in the homes of 75 control subjects (relative risk 7.0) and identified a clear dose-response relationship. The asthmatic subjects tended to live in older, more humid housing (180). In a survey of the mite fauna in 30 homes in Oxfordshire, bedroom relative humidity was the most important factor related to the number of mites found in the mattress. Lack of heating and therefore lower bedroom temperatures were associated with higher mite counts (95). Kuehr *et al* studied the homes of 1050 children in Germany and found *Der p I* levels to be associated with low storey level, damp in the bedroom, higher relative humidity and use of a blanket of animal hair or old mattress (181). Van Strien *et al* measured *Der p I* levels in floor dust and mattress dust in 516 homes in the Netherlands (99). Higher allergen levels were associated with carpeted floors, increasing age of dwelling and with increasing number of occupants. *Der p I* concentrations were positively associated with average relative humidity in the bedroom over a 3-6 week period, with a tendency towards higher allergen levels in homes with reported or observed signs of dampness. A significant correlation was seen between *Der p I* levels in different locations within the home.

In a case-control study in the Stockholm area of Sweden (182), house dust mite

sensitive children were more likely to have lived in areas infested with mites than control children. Sensitisation to mites was related to indicators of damp in the home and higher levels of mite allergen occurred in homes where dampness was perceived as a problem. They postulated that mite infestation of homes was becoming more prevalent in Sweden and may be related to energy saving measures creating more optimal conditions for house dust mite survival. These findings were confirmed by Munir *et al* who investigated the levels of mite allergen in dust from the homes of asthmatic children from three climate zones in Sweden (183). Mite allergen levels were confirmed to be higher in homes where damp was a problem and in homes with higher absolute indoor humidity and poorer ventilation.

As part of the European Community Respiratory Health Survey, another Swedish group studied the relationship between the indoor environment and asthma like symptoms (96). The majority of the houses in the study had a relative humidity higher than 45%, which is recognised as a threshold for mite viability. Despite this, the prevalence of house dust mite was very low at 13% homes surveyed. Their presence was associated with higher indoor humidity and increasing age of the house and was identified as an independent risk factor for asthma related symptoms, odds ratio 7.9. Levels of indoor mould were related positively to humidity, but no comment was made on any association with asthma like symptoms.

A study of HDM allergen level in 108 French homes found that homes with low relative humidity had low mite-allergen content. However, homes with intermediate relative humidity levels had very variable mite allergen content suggesting other factors also influence mite infestation (184).

Mite allergen levels in house dust are also dependent on their rate of degradation and removal. Under laboratory conditions mite allergens are extremely stable and can persist for up to 18 months at temperatures and humidities found in normal domestic circumstances (185). Mite allergen exposure is therefore likely to be largely dependent on both mite numbers, controlled by environmental conditions, and removal of allergen through repeated vacuuming and cleaning.

1.15.3 House dust mite avoidance measures

House dust mite avoidance improves asthma symptoms and reduces non-specific bronchial reactivity. It was previously the practise to send patients with severe

asthma to sanatoria at high altitudes. Such locations have cold, dry climates, inhospitable to the house dust mite (93-94, 186-187). Similarly, the admission of patients to hospitals where there are few mites improves asthma symptoms. These observations prompted investigations as to whether a reduction in mite allergen exposure was possible within the home.

The effects of changes in house dust mite allergen exposure on asthma severity were investigated by Marks *et al* in 34 house dust mite sensitive subjects with asthma (188). Changes in allergen concentration in the subject's bed were significantly correlated with a reduction in airway reactivity and improved asthma symptoms. The magnitude of these changes was fairly modest but the results suggest that reduction of house dust mite allergen, particularly in the bed may improve asthma control in mite sensitive subjects.

Other studies have also documented a modest improvement in asthma control using such measures as plastic coverings, frequent Hoovering or use of acaricides and liquid nitrogen on mattresses to kill mites (189). Such mite avoidance measures are not universally practised as the benefit is often not immediately apparent. Economic factors may influence the utilisation of such techniques with fewer asthmatics from lower socio-economic classes using allergen avoidance measures (190).

It is estimated that mite numbers have to be reduced ten fold to significantly improve asthma symptoms and local measures to control mite numbers are therefore unlikely to be successful. Some authors have advocated controlling mite numbers by altering the indoor environment either by reducing relative humidity or increasing ventilation. A Danish group investigated 30 asthmatic patients before and after moving to homes with mechanical ventilation systems (191). The house dust mite numbers were significantly lower, even after 15 months in the new home. This was thought to be due primarily to a reduction in the indoor air humidity, as resident indoor behaviour remained relatively unchanged throughout the study period. The same group also found a reduction in asthma symptoms and medication requirements in fourteen house dust mite sensitive patients moved to purpose built healthy homes. This was attributed to lower levels of HDM exposure (192).

In a recent British study (193), house dust mite counts and allergen levels were measured in six houses supplied with a portable dehumidifier and in six control houses. Although condensation was decreased in the dehumidifier group, indoor

relative humidity was not consistently reduced to the level required to retard mite growth and no differences in house dust mite numbers or allergen levels were identified. However, the number of houses studied was small and the dehumidifier was placed at a central location within the house. It is reported that ventilation of the bedroom is as important as ventilation of the rest of the house in controlling HDM infestation (178). It is possible that a dehumidifier placed in a bedroom could significantly reduce mite numbers in the mattress and bedroom floor, which may be of potential benefit to an asthmatic individual.

1.16 Storage mites and asthma

Although *D. Pteronyssinus* is the commonest house dust mite in Britain, other mites are present in house dust, usually in smaller numbers (97). Some hydrophilic species such as the storage mites require higher relative humidities for optimum growth. They were first identified in rural communities in dwellings with high moisture content, often in grain, hay and straw. Exposure to these storage mites can cause symptoms of bronchial asthma, allergic rhinitis and conjunctivitis (194) and is an occupational risk among farmers (195).

There is also evidence of sensitisation to storage mites in urban populations. In 1967, it was suggested that storage mite allergy may be important in countries with a damp climate (196). In 1979 Wraith *et al* showed an association between dampness in the dwelling and a positive skin prick test to storage mites (197). In 1990 Iversen *et al* found storage mites in significant numbers in house dust from damp dwellings (98). They concluded that allergy to storage mites in farmers might be partly attributable to indoor exposure as a consequence of living in damp housing conditions (195). Luczynska *et al* found a 14% IgE response to one of three storage mites in factory workers not occupationally exposed to storage mites (198). It was suggested this may be due to allergenic cross-reactivity with the house dust mite but this has subsequently been found to be incorrect (199).

1.17 Moulds

Fungi are a heterogeneous group of non-photosynthetic plant bodies. The minimum relative humidity permitting growth varies between 75 - 95% for different mould species and the optimum temperature for growth is 20 - 40°C. The fungal propagules

or spores are a major factor causing allergy. Spores range in size from 3 - 200µm but most are between 5 - 10µm in diameter and within the respirable range. Although pollen has been widely investigated as an aeroallergen, less is known about fungal spores, which are present in outdoor air in far greater numbers than pollen grains. Large numbers of fungal species are uncharacterised and most studies of fungi as aeroallergens have been limited to the class Fungi Imperfecta which includes *Cladosporium*, *Alternaria* and *Aspergillus* species (101).

Fungi are common contaminants of indoor air and dust (200-201). They can originate from outdoor sources or from growth on moist substrates within the home. Conditions in the home are crucial determinants of the airflora. Moulds thrive in moist conditions, with a suitable substrate and moderate temperatures. Where a house is well ventilated, the number of propagules and diversity of fungi in indoor air reflects that of the outdoor air. New construction techniques aimed at energy conservation often lead to an increase in indoor humidity and lower air exchange rates. This combination predisposes to condensation which tends to increase the number of indoor moulds.

1.17.1 Indoor Moulds

Fungal propagules are a major component of house dust (201-204). Indoor airborne levels reflect outdoor levels, the presence of indoor mould growth and physical activity within the dwelling. The presence of moulds in indoor air is only weakly related to house damp and the presence of indoor mould (205). Estimates of potential exposure to fungal propagules vary widely. Direct sedimentation of spores on exposed plates is the least accurate, seriously underestimating spores of small size. Other sampling methods used include culturing airborne propagules or direct plating or culturing of house dust. Different analytical methods can give vastly different results and single measurements do not provide a reliable measure of exposure to fungi in indoor environments (206).

Despite these limitations, most studies agree on the mould species found indoors, although different geographical location, season of sampling and type of building influence the quantity in which they are found. In British and other north European studies, the predominant species are *Penicillium* and *Cladosporium* with lower levels of *Alternaria* and *Aspergillus* (200-201, 207-208).

1.17.2 Mould allergy

Feinberg in 1935 was one of the first to acknowledge the potential importance of moulds as allergens to the asthmatic. He found in a group of 243 patients with respiratory allergy, 68 (28%) gave positive skin prick reactions to one or more mould extracts. Hendrick 40 years later analysed skin prick test reactions to 22 common allergens including eight moulds, in 656 asthmatic patients (209). Thirteen percent had positive reactions to *Aspergillus fumigatus* and 18% reacted to other moulds.

Mould allergen extracts are extremely variable and complex, making it difficult to produce standardised mould allergen extracts for skin prick testing. Hence the prevalence of skin prick test diagnosed mould allergy can vary considerably with both the number and quality of the mould extracts used for testing. The extent of cross-allergenicity among common airborne fungal spores also remains to be resolved. Furthermore, atmospheric fungal spore concentrations do not necessarily reflect the types of fungi which most frequently induce sensitisation, indicating that moulds have different sensitising capacities (210). *Aspergillus* in particular shows a high sensitisation rate in comparison with its low airborne concentrations. Atopic subjects will usually have multiple skin prick reactions to other common allergens such as house dust mite, pollens and animal dander (209). The clinical relevance of mould allergy is therefore difficult to isolate, especially as there is no well defined mould season during which patients could be studied.

1.17.3 Moulds and asthma

In 1873 Charles Blackley reported that after breathing cultures of *Penicillium glaucum* he experienced "bronchial catarrh which almost unfitted me for duty" and commented that the reaction had been so severe he had no wish to repeat the experiment (211). Some 50 years later, Chadham reported three cases of asthma induced by *Puccinia graminis* in workers in wheat fields (212). All were skin test positive to this mould and exposure to a few spores caused acute bronchospasm. Hopkins *et al* also reported a case of asthma induced by the mould *Alternaria* isolated from the patients cellar (213).

One of the first case-control studies investigating the association between moulds and asthma was performed by Sherman in 1964 (214). He identified 38 patients,

clinically sensitive to the mould *Alternaria*. On surveying their homes, moulds were present in virtually every case, *Penicillium* and *Aspergillus* being the commonest species identified. In the homes of 24 control subjects, moulds were found in only nine (38%) homes. The asthmatic patients in whose homes the mould *Alternaria* was identified did not have more symptoms than the other *Alternaria* sensitive patients. They concluded that a positive skin test to a mould did not necessarily indicate clinical sensitivity to it.

In a more recent case-control study from Britain (208), 72 asthmatic patients identified from general practise lists and 72 age and sex matched controls were studied. Positive skin tests were found to one of five moulds in nine asthmatic patients and one control. Visible mould growth was reported in the homes of 19 (26%) asthmatics and 9 (12.5%) controls, although the number reporting damp patches within the home was similar for both groups (asthmatics 39%, controls 37.5%). Again the commonest mould identified in the home was *Penicillium* and *Penicillium* IgE (RAST) antibodies were frequently found in these patients.

Although the presence of mould in the home is associated with respiratory symptoms and asthma, attempts to correlate objective measures of mould quantity with respiratory symptoms have met with varied success. Platt *et al* (146) demonstrated a relationship between wheezing in children and airborne mould spore concentrations, but Strachan (149) found only a small, non-significant increase in total airborne mould counts in the homes of children with exercise induced bronchoconstriction. In a small study of eight asthmatic, mould sensitive patients (215), significantly lower peak flow values were found on days with the highest concentrations of outdoor airborne fungi but changes in indoor airborne mould levels had little influence on peak flow rates or respiratory symptoms. High outdoor fungal spore concentrations were associated with asthma symptom severity and medication use in a small group of asthmatic children (38).

A large study on the Isle of Wight skin prick tested 981 four year old children to a battery of common allergens (216). Six per cent of children had positive skin prick tests to *Alternaria* and *Cladosporium*, the third most common cause of sensitisation after house dust mite and grass pollens. Mould sensitivity correlated with diagnoses of asthma, eczema and rhinitis. No associations was found between mould sensitivity and age of the house, exposure to pets, passive tobacco smoking or season of birth.

1.17.4 Fungal volatiles and mycotoxins

It has more recently been suggested that the health of occupants of mould affected houses is affected by fungal volatiles. These comprise complex mixtures of alcohols, aldehydes, esters and aromatics which account for mouldy odours. Symptoms such as eye, nose and throat irritation have been attributed to inhalation of these volatiles from mould growths in buildings in the Netherlands, Sweden and Canada. Inhalation may cause acute respiratory responses including wheeze (217) and the effects of prolonged exposure are unknown.

Mycotoxins are complex organic chemicals associated with the spores of certain moulds. These mycotoxins can cause illness in humans and animals, usually as a result of ingestion from contaminated food. Little is known about toxicity resulting from inhalation, but as the majority of mould spores are within the respirable range, high concentrations of mycotoxins could reach the alveoli where they will be readily absorbed because of their low molecular weight and solubility. Once absorbed their systemic effects would be similar to those of mycotoxins absorbed by other routes.

1.18 Bacteria and micro-organisms in house dust

There is very little published work on airborne bacteria in houses. Investigations of bacteria in buildings have largely been due to the investigation of humidifier fever or sick building syndrome. However, a Swedish study recently reported that increased levels of airborne bacteria may be associated with asthma (96). The homes of subjects with asthma related symptoms had significantly higher total levels of bacteria and mould and a higher proportion had detectable levels of house dust mite allergen compared with the homes of subjects with no asthma related symptoms. These associations persisted after controlling for smoking, indoor temperature and air humidity. Inhaled bacterial particles may cause an inflammatory reaction in the bronchi, even without an infection, resulting in a deterioration in asthma symptoms.

Chapter 2

Indications for present research

2.1 Indications for present research

There is little doubt that the prevalence of asthma is rising in industrialised countries. This is thought to reflect some adverse change in the environment, as it is unlikely the gene pool could have changed so rapidly as to increase the number of individuals genetically predisposed to allergic diseases. Attention has therefore focused on the environment and in particular changes in the indoor environment occurring in recent years, which could explain the increase in asthma prevalence.

Damp housing is known to be a common problem on housing estates in central Glasgow. Prior to this study, patients with asthma attending the Southern General Hospital Chest Clinic would occasionally ask for a medical report to support their application for rehousing, on the grounds that the damp in their home had an adverse affect on their asthma. This prompted a review of the literature and it became evident that further research was required.

Many previous cross-sectional epidemiological studies reported a higher prevalence of respiratory symptoms, including those common to asthma, in subjects living in damp or mouldy housing. However, as asthma is largely a clinical diagnosis and a satisfactory definition for epidemiological purposes remains elusive, it is difficult to identify subjects with asthma in such surveys.

There are few case-control studies designed to investigate the relationship between damp housing and asthma. None were conducted in Britain, indeed two were conducted in Africa where climate and housing conditions vary greatly from those found in Britain. The majority relied on measures of self-reported damp and are therefore susceptible to respondent and recall biases. Furthermore, none of them quantified the severity of damp or mould in the dwelling or the severity of asthma. As such it has not been possible to demonstrate a dose-response relationship between severity of damp or mould in the dwelling and the severity of asthma.

The main objectives of this thesis were two-fold:

1. By using a case-control study design and an independent surveyor to establish the presence and severity of damp housing conditions, the principal study aim was to identify if subjects with physician diagnosed asthma were more likely than age and sex matched controls to live in damp or mouldy homes.

2. To establish any association between asthma severity and severity of damp and mould in the home, in effect whether there was a dose-response relationship.

Chapter 3

Methodology

3.1 Subjects recruited

3.1.1 Asthmatic subjects

In a case-control study consideration must be given to the appropriate selection of the cases. In attempting to determine if there is an association between damp housing and asthma it could be argued that it would be better to use incident (new) cases of asthma rather than prevalent (existing) cases with varying disease duration. The time relationship between the onset of asthma and exposure to damp housing conditions may be clearer in newly diagnosed cases rather than in long-established cases. However, when assessing whether damp housing conditions adversely influence asthma severity, prevalent cases may be preferable to incident cases as chronic asthma severity is difficult to measure in the latter.

As asthma is generally first diagnosed and managed by a subject's general practitioner, incident cases would have best recruited from the primary care setting. With no "gold standard" diagnosis of asthma, problems were anticipated in obtaining precise criteria for eligibility for entry to the study. It was decided that patients with existing (prevalent) physician diagnosed asthma, attending a hospital out-patient clinic would be studied. By selecting such patients, the results of the study would not be representative of all cases of asthma, as there would be an under-representation of those with well controlled or mild symptoms who would be managed entirely in the primary care setting.

Asthmatic subjects were recruited from the chest clinic at the Southern General Hospital. This is a District General Hospital situated on the southern bank of the river Clyde in Glasgow and provides respiratory health care for a population of approximately 200,000 living within the South-West area of the city, predominantly within postal code areas G51, G52 and G53. Consecutive patients aged between 5 - 44 years, with physician diagnosed asthma, who attended either the paediatric or adult asthma clinics between November 1992 and February 1993, were eligible for entry to the study. Three subjects refused to participate leaving 102 patients with asthma (cases) to be entered.

3.1.2 Control subjects

The purpose of the control group in this study was to provide an estimate of the

expected exposure to damp housing if there were no association between damp housing and asthma. Ideally cases and controls should be drawn from the same source population.

Patients attending a hospital clinic with any chronic illness are likely to differ from a random sample of the population. The selection process of seeking medical care and differing referral practices of general practitioners is likely to bias the sample. It is frequently difficult to identify the sub-population from which comparable controls should be drawn. In this instance there were two alternatives; either the group of control subjects could be randomly selected from 1) another hospital out-patient clinic or 2) the general population.

A control group of patients with some other chronic non-respiratory illness could have been selected from another clinic within the hospital. There were however disadvantages to this. The probability of reaching a hospital out-patient clinic is likely to vary for different diseases. For example, general practitioners are likely to refer the majority of their younger patients with diabetes mellitus to a hospital specialist at the time of diagnosis, whereas only patients with moderate to severe asthma are likely to be referred to a hospital asthma clinic. Controls from another hospital clinic would therefore represent a different selected group of the general population. Any differences between the cases and the controls could have been more related to the epidemiology of the disease of the control subjects rather than with the disease under study.

Secondly due to the age range of the cases studied, there were difficulties in identifying controls of similar ages attending another clinic within the Southern General Hospital. Asthmatic children were recruited from the paediatric asthma clinic but there was no other suitable paediatric clinic within the hospital, from which controls could be selected.

It seemed more practical to select control subjects from the general population living within the catchment area of the Southern General Hospital. By recruiting the control subjects from the same residential area of the city (postal code areas G51, G52 and G53), they would be more likely to be of comparable socio-economic status to that of the asthmatic subjects. Any differences between the cases and controls would, as far as possible, be controlled for in the statistical analysis of the data.

For each asthmatic patient entered, two control subjects, matched for gender and age to within five years, were randomly selected from the Greater Glasgow Health Board Community Health Index. Attempts were then made to contact the control subjects and obtain their consent for entry to the study. If the control subject was either no longer resident at the contact address or refused to participate, another matched control was selected at random from the Health Index. The ratio of controls to cases was maintained at approximately 2 to 1. In all, 201 of the 450 subjects randomly selected from the index were no longer resident at the contact address and could not be traced. Of the 249 subjects successfully contacted, 196 (79%) agreed to participate. All asthmatic and control subjects lived within the catchment area of the hospital defined by Glasgow area postal codes G51, G52 and G53.

The prevalence of asthma in the general population is estimated between 10-15% (20). It was anticipated that a similar proportion of control subjects would have respiratory symptoms consistent with asthma. Any control subject with established physician diagnosed asthma would be excluded and an upper age limit of 45 years was selected to minimise the number of subjects with smoking related symptoms of airflow obstruction. It was not possible for a hospital physician to formally screen all control subjects for asthma and all control subjects were retained in the main analysis of the data. If the control group were to contain a small number of asthmatic subjects this would bias any odds ratios towards unity and would increase the risk of a type II statistical error. A sub-analysis of the data was planned, to exclude non-smoking control subjects who admitted to intermittent respiratory symptoms of wheeze and chest tightness, so that the magnitude of this bias could be assessed.

3.2 Questionnaire

3.2.1 Socio-economic data

The questionnaire (Appendix A) was a modified version of that used by Martin *et al* (145) in two previous studies designed to investigate the relationship between damp housing and prevalence of respiratory symptoms. Few problems had been encountered with this questionnaire. It gave information on the number and ages of all residents in the dwelling; the number and type of rooms in the dwelling; the presence and type of any pets; smoking status of all adults and children (life-long non-smoker, ex-smoker, current smoker or passive smoker); type of heating (central heating, gas or electric fires, paraffin heaters), cooking facilities (gas or electric

oven); washing and drying facilities (frequency of washing and method of drying of clothes); occupation of all adults in the household (employed, unemployed adult subject or parent of child subject, no adult in paid employment in the household) and net weekly household income.

3.2.2 Measures of self-reported damp

Exposure to damp housing conditions was assessed from questionnaire responses. The respondent reported the presence of damp or condensation in their current dwelling and graded this as either a serious problem or more of a nuisance than a problem. The presence of rooms in the home which they were either reluctant or unable to use because of damp or cold was also recorded. Further questions enquired about exposure to damp and mould in their previous dwelling and whether this had contributed to their moving house.

3.2.3 Respiratory health questions

Seventeen questions considered to give information on the physiological severity of asthma were selected from other respiratory questionnaires and piloted on 25 asthmatic subjects attending the asthma clinic, prior to commencement of the study. Eleven questions were selected as giving information as to the severity of an individual's asthma that was consistent with clinical judgement and were included in the main questionnaire. These questions related mainly to symptoms such as frequency of wheeze, cough and chest tightness; degree of dyspnoea on exercise; current asthma medication requirements; and use of oral prednisolone within the previous 12 months. Questions regarding functional asthma severity status were included, such as number of days off work, school or spent indoors with asthma in the previous 12 months; whether adult subjects were regarded as sick or disabled for employment purposes; and the subjects own perception of their health during the two weeks before interview, graded on a five point scale from excellent to very poor.

3.2.4 Administration of questionnaire

All asthmatic and control subjects were asked to participate in a health survey

and were not informed of the purpose of the study prior to completion of the questionnaire. Each subject completed a structured interview, usually in their home, with a trained researcher who was blind to their health status, using the questionnaire described above. A small group of interviewers were briefed and became familiar with the questionnaire before beginning the study. This method aimed to standardise the administration of the questionnaire and maximise the amount of data obtained, thus minimising the possibility of non-response bias. If the subject was a child, wherever possible the parent or guardian answered the questions concerning the child's health.

3.3 Lung function

Patients performed spirometry (Vitalograph) at the asthma clinic at entry to the study. This was performed to similar guidelines published by the British Thoracic Society except that no measurement of relaxed vital capacity (VC) was made and the procedure for the forced vital capacity (FVC) was terminated after six seconds. Some of the younger children were unable to perform the measurements adequately. The forced expiratory volume in one second (FEV_1) and the ratio of FEV_1 to the FVC were recorded. FEV_1 and FVC were expressed as percentages of predicted values. The prior use of bronchodilators was not recorded.

3.4 Skin testing

Skin prick allergy testing was performed on consenting asthmatic and control subjects to determine atopic status and prevalence of allergy to common indoor moulds. All allergy testing was performed by a specialist nurse according to the technique described by Pepys (218). Subjects were requested not to take any anti-histamine preparations for 48 hours before the tests. Allergy solutions were supplied by Bencard Ltd and stored as recommended at 4°C.

A drop of each allergen and a negative control were applied to the volar surface of the forearm. A sterile lancet was advanced through each drop of allergen, the epidermis gently raised and the lancet withdrawn. Results were read 15 minutes after application and recorded as the mean of the largest diameter and the perpendicular diameter of the wheal. Any erythema or flare was ignored. A mean

wheal diameter of 3mm greater than the control was regarded as a positive reaction. A subject was classified as atopic if they had one or more positive reactions to either house dust or house dust mites; mixed pollens; cat or dog dander.

Nine common indoor moulds were used to assess mould sensitivity. These were *Cladosporium*, *Alternaria*, *Penicillium*, *Aspergillus fumigatus*, *Aspergillus niger*, *Fusarium*, *Rhizopus*, *Sporobolomyces* and *dry rot*. Subjects with a positive reaction to any of the moulds were regarded as mould sensitive.

3.5 Measurement of asthma severity

3.5.1 Quantifying severity of chronic asthma

Although it is intuitively clear that diseases such as asthma vary in severity, measurement of this severity poses methodological difficulties. Three categories of disease severity are often measured, namely physiological severity, functional severity and burden of illness (219). Physiological severity reflects the interaction between biological severity and environmental factors, including medical treatment. In asthma, this may include the number of acute attacks, the number of hospital admissions, or the degree of lung impairment. Functional severity on the other hand relates more to the ability to perform activities of daily living and assesses disability and quality of life. The burden of illness refers to the impact of the disease on family or society and formal measurements of this were beyond the scope of this study.

The method of recording disease severity is likely to influence the resulting measurement. Whilst clinicians are more likely to target physiological severity, families may focus on burden of illness and the patient on functional status. Whatever measures of illness severity are recorded, they cannot be objectively validated against a “gold standard”. It is preferable to try and validate one measure of disease severity against another. Obtaining a measure of asthma severity is further complicated by fluctuating symptoms and lung function characteristic of the disease. A severity measurement which took this into account would be an advantage.

3.5.2 Physiological severity of asthma

Measures of the physiological severity of asthma are normally based on a

combination of clinical criteria such as symptoms and treatment requirements, objective measurements of lung function and bronchial hyperreactivity. Several severity scoring systems have used some or all of these criteria (54, 160, 220). These were all designed for use within a specified population of asthmatic subjects and none were applicable to the asthmatic subjects selected in this study. My aim was to develop an asthma severity scoring system which could be used to determine if damp housing conditions were more prevalent in patients with severe compared with mild asthma and to investigate any relationship between quantity of damp or mould in the home and severity of asthma.

3.5.3 Development of a chronic asthma severity score

An asthma severity score was calculated for each patient based on the questions relating to the physiological severity of asthma (table 4). The scoring system was similar to that adopted by the American Thoracic Society in its guidelines on the evaluation of impairment /disability in patients with asthma (221). Scores were allocated for the frequency of asthma symptoms, degree of dyspnoea on exercise, frequency of use of their relief inhaler, use of nebulised drugs, current inhaled steroid requirements and use of oral steroids. The FEV₁ measured at the last clinic visit was also included in the scoring system. The sum of these components of the scale comprised the asthma severity raw score, with a possible range of 0 - 24. Measures of the reliability and validity of the asthma severity scale were made using standard methods of scale development (222).

Table 4. Asthma severity item scores

Severity Item	Score	n
Frequency Asthma Symptoms		
Never	0	0
Less once per week	1	29
2 - 3 times per week	2	28
4 - 5 times per week	3	9
Most days or nights	4	36
Shortness Of Breath On Exercise		
Never	0	8
Playing games or sports	1	4
Walking uphill or stairs	2	39
Walking on level	3	15
Walking around house or dressing	4	36
Inhaled B₂ Agonist Use		
Never	0	2
Less than daily	1	35
Daily	2	65
Dose Inhaled Steroid		
None	0	9
Up to 400µg daily (or Intal)	1	25
401 to 800µg daily	2	15
801 to 1500µg daily	3	35
greater than 1500µg daily	4	18
Nebuliser Use		
No	0	84
Yes	2	18
Use Oral Steroids Last 12 Mths		
None	0	44
1 or 2 short courses	1	29
more than 2 courses	2	25
Maintenance	4	4
Lung Function : FEV₁ (%predicted)		
Greater than 80%	0	57
70 - 79%	1	11
60 - 69%	2	14
50 - 59%	3	6
less than 50%	4	9
Not measured	-	5

3.5.4 What is the scale measuring?

The underlying phenomenon that a scale is intended to reflect is often referred to as the latent variable or factor. In this study a scale was required to measure chronic asthma severity. The responses to questions thought to give information on severity of asthma were selected as items for inclusion in the severity scale. A principle components factor analysis of the scale items was then performed. Principal components analysis is a mathematical technique that allows the determination of the number of latent variables or factors underlying a set of items. Although it was anticipated that the scale described above would have only one latent variable (asthma severity), principal components analysis was used to check this assumption was correct.

The number of underlying factors extracted from a set of scale items by the analysis will depend upon the extraction criteria used. The method of factor extraction employed in the development of the asthma severity scale was based on Cattell's scree test. Cattell recommended plotting the variance explained by each successive factor (223). As each factor explains less variance than the preceding factors, a line connecting the plots of variance explained by each factor will run from the top left to bottom right of the resultant scree plot. An "elbow" in the plot represents the point below which factors explain relatively little variance in the scale items (likened to the debris or scree at the bottom of a mountain side). Cattell's guidelines suggest retaining factors above the elbow and rejecting those below it, that is retaining the factors that contribute most to the explanation of variance in the total set of original items. The factor scree plot in figure 1 shows a clear "elbow" cut off point and as anticipated indicates the presence of only one factor (physiological asthma severity) contributing significantly to the explanation of variance in the original asthma severity scale items.

Principal components analysis also checks how the original scale items load onto the extracted factor(s). In the scale of asthma severity, all severity item factor weights were relatively high, indicating they all contributed significantly to the underlying factor of asthma severity, but those concerning medication requirements in the form of daily dose of inhaled steroid and use of oral prednisolone loaded more strongly than the other variables (table 5).

Figure 1. Principal components factor scree plot

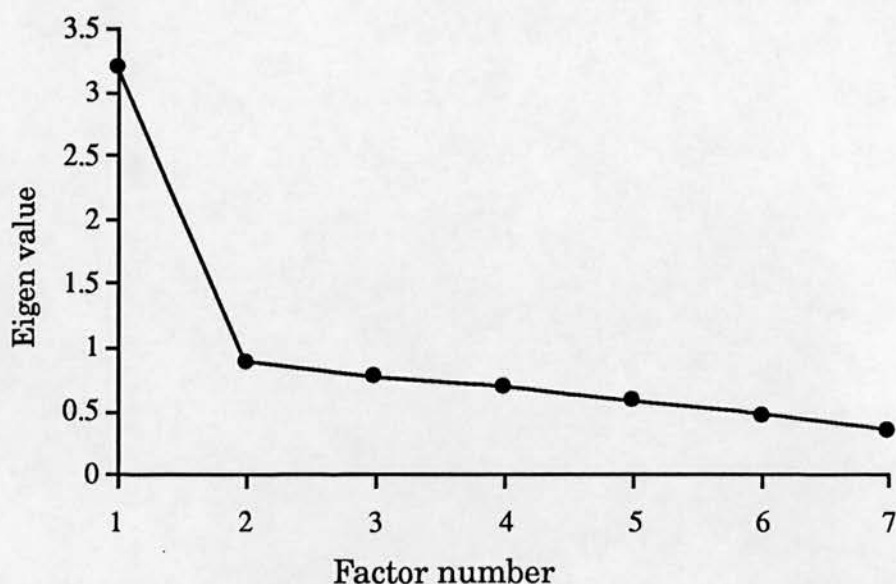


Table 5. Principal components factor analysis of asthma severity score

Variable	Factor Weight
Frequency of symptoms	0.68
Dyspnoea on exercise	0.71
Relief inhaler use	0.62
Inhaled steroid use	0.72
Oral steroid requirements	0.77
Nebuliser use	0.56
FEV ₁ (% predicted)	0.67
Eigenvalue	3.23
% variance	46.2%
Cronbach's alpha	0.79

The use of principal components analysis in the development of the asthma severity score confirmed the assumption that there was a single underlying dimension of physiological asthma severity that could be measured and that the original scale items grouped in a consistent manner. Confirming that the severity scale measured what it was designed to do, further assessments about reliability were made.

3.5.5 Scale reliability

Scale reliability refers to its ability to produce consistent results. The use of composite scales of measurement based on a number of related items, such as those proposed to measure asthma severity, generally increases reliability. Measures of internal reliability are concerned with the homogeneity of the items comprising the scale. If the scale items have a strong relationship to the underlying measurement, in this case asthma severity, then they will also have a strong relationship to each other. A scale is said to be internally consistent to the extent that its items are highly inter-correlated. A uni-dimensional scale such as that proposed to measure physiological asthma severity should consist of a set of items that correlate well.

The internal reliability of the asthma severity score was assessed using Cronbach's alpha coefficient. This statistic gives an indication of the degree of inter-correlation between the scale items (224). A Cronbach's alpha greater than 0.70 confirms a good degree of internal reliability. A value greater than 0.80 and the scale is consistent, with the sum of the item responses yielding a score representing the underlying dimension of the individual items. Cronbach's alpha coefficient for the seven item asthma severity score described above was 0.79 indicating a high degree of internal consistency.

A measurement scale can also be described as consistent if similar results are obtained when it is administered to the same population on different occasions. This test-retest reliability of the asthma severity score was not assessed in this study.

3.5.6 Scale validity

Validity refers to the extent to which the scale measures the underlying concept studied. As the original scale items in this study all represent different aspects of physiological asthma severity, the final scale would also be expected to provide a measure of asthma severity, that is to have face validity. Further, the original scale items included different aspects of physiological severity of asthma including symptom frequency, treatment requirements and lung function measurements so that the final scale has content validity.

The main difficulty arises when attempting to establish criterion validity, where the scale would be required to be compared to a "gold standard" measurement of asthma severity. As the development of the proposed asthma severity score was precipitated because there is no current well validated measure of asthma severity, criterion validity cannot be established. Another method of appraising validity is to identify if the scale measurement relates to other variables that should be linked to the characteristic under study. If this were the case, then the scale would have construct validity.

To give an indication of the construct validity of the asthma severity scale, correlations were sought between the raw severity scores and indices of functional asthma severity and burden of illness, as assessed by questionnaire responses. The raw severity scores were statistically correlated with the patients own perception of their health over the two weeks prior to interview, graded subjectively on a five point scale from excellent to very poor ($r_s=0.48$, $p=0.0001$); the number of days off work, school or spent indoors because of their asthma in the last twelve months ($r_s=0.55$, $p=0.0001$); if adults with asthma were regarded as sick or disabled for employment purposes ($p=0.0001$); and were negatively correlated with net household income ($r_s= -0.34$, $p=0.001$). These findings would support the case for the severity scale having construct validity.

3.5.7 Ordinal scale of asthma severity

The above results suggest the asthma severity raw scores could be used for statistical analysis as a continuous variable to give a reliable indication of the physiological severity of a patient's asthma. It does not lend itself to interpretation in a clinical setting. In an attempt to do this, the raw score values were collapsed to an ordinal scale representing mild (raw score between 0 and 8), moderate (between 9 and 13) and severe (between 14 and 24) asthma. The cut-off at appropriate raw score values for these asthma severity groups were chosen arbitrarily such that the percentages in each severity group were equal. Checks were then made to identify if patients allocated to a particular severity group displayed the appropriate expected clinical characteristics (table 6).

Table 6.**Grading of asthma severity.**

Figures represent number(percentage)

Asthma severity grade	Daily Symptoms	Daily dose inhaled steroid > 800µg	FEV1 < 80% predicted	Adults sick or disabled
Mild	2 (6)	4 (12)	1 (3)	1 (5)
Moderate	8 (25)	17 (53)	12 (37)	8 (32)
Severe	24 (75)	30 (94)	27 (84)	23 (77)

Two patients allocated to the mild severity group had daily asthma symptoms and may have been under-treated. The majority of those in the severe asthma group had daily asthma symptoms and greater degrees of airflow obstruction, despite appropriate treatment with high dose inhaled corticosteroids. Two subjects classified as having mild or moderate asthma were on high dose inhaled steroids (step 4 British Thoracic Society treatment guidelines) and may have been over-treated. There was an appropriate increase in the number of adult patients unable to work because of their asthma in the higher severity groups. The grading of asthma severity, using the proposed ordinal scale, appeared to be consistent with clinical expectation.

3.6 Methods of assessing damp in dwellings

3.6.1 Self-reported or objective measurement of damp?

There is no standard method of assessing the exposure of an individual to damp or mould in the home. Dampness can be described in various ways. If asked during a questionnaire a householder may describe damp as the presence of mould growth on walls or excessive window-pane condensation within the dwelling. A surveyor on the other hand may describe damp as a positive reading on an electronic damp meter. Although there is some degree of correlation between these different means of describing damp, it is known from previous studies that measures of self-reported damp are unreliable and that objective measures of damp are desirable.

Materials within dwellings do not become visibly damp and may not even feel damp to the touch until they are severely affected. Dampness may be hazardous to

materials long before it can be detected by the unaided senses, but whether this is also the case with respect to an individual's health is unknown. A sensitive measure of assessing damp in buildings is therefore required.

3.6.2 Moisture content of building materials

The percent moisture content of a building material is difficult and inconvenient to measure as it is necessary to take samples of the material for processing in a laboratory. It is also not a particularly meaningful measurement in determining whether a material is wet or dry. For instance, wood may have a moisture content of 12% and be described as dry whereas plaster may be wet at a moisture content of 1%. A more important measure of damp is the excess free moisture within a material, which will be in a state of dynamic equilibrium with the surrounding environment. The free moisture, available to support mould growth for instance, will be continually changing, dependent on ambient temperature and relative humidity. Localised variations will occur with time and location within the dwelling. Meaningful assessments of damp should take these factors into account.

3.6.3 Theoretical ideal of damp measurement

The theoretical ideal would be to cover the suspect damp area of wall with a waterproof box, enclosing a humidity measuring device. Free water evaporating from the wall will raise the RH of the air within the box until equilibrium is reached. The resultant RH measurement would indicate how damp the wall is, regardless of the ambient RH in the room. This process however is too laborious for survey purposes as it takes several hours for the RH to reach equilibrium at each survey point. However, if the volume of air around the RH measuring device were to be reduced to a minimum, equilibrium times would be correspondingly reduced, perhaps to a level when the technique could be used for routine survey purposes.

3.6.4 Can this theoretical ideal be used in damp surveys?

This possibility was investigated by McGill (personal communication). He used a rubber hemisphere to entrap a small volume of air (6mls) around a digital hygrometer probe applied to the surface of a material. For calibration purposes the

materials used were small pieces of softwood and plasterboard climatized in sealed units with RH levels ranging from 66 to 100% at an ambient temperature of 20°C. Measurements of RH at the sample surface were taken after 1 and 5 minutes. As the entrapped air approaches equilibrium with the free moisture in the sample, the RH measurements should approximate to the humidity in which the sample was climatized. The RH measurements taken after 1 minute were approximately 10% below, whereas those taken after 5 minutes were very close to the theoretical ideal. Equilibrium of free moisture occurred between one and five minutes (225).

Compared with more traditional damp survey methodology, the above method of assessing damp significantly increases the overall time required for a house dampness survey. It was thought it may be practical for surveying the occasional dwelling, but not for surveying a large number of dwellings over a short time period as was proposed in this study.

3.6.5 Use of a damp meter in detecting and quantifying damp

Most dry building materials form an insulator and will not conduct electricity. If water is added, minute amounts of salts within the material are dissolved and the material will begin to conduct electricity. The measure of this electrical conductance is proportional to the amount of free water within a material, that is water which is free to produce the effects on the material usually associated with dampness.

A conductance type electronic moisture meter quantifies the free water in a material by measuring the amount of current which passes between two sharpened steel electrodes pushed into the building material of interest. The more free water within the material, the better current will be conducted. The percentage scale deflection on the meter therefore gives an indication of the relative dampness of the material. A high reading on such a meter (in the absence of contaminating salts which can give spuriously high readings) indicates a damp condition of approximately equal significance in wood, brick, plaster or wallboard, regardless of their very different total moisture contents (226).

Moisture meters have been used by surveyors when assessing damp in dwellings since 1956. They are widely regarded as reliable and can detect hazardous damp long before it can be detected by the human senses. This rapid semi-quantitative method of assessing damp in dwellings was felt to be appropriate for use in this study.

3.7 Survey of dwellings

After completing the questionnaire, subjects were asked if their home could be independently assessed for damp and mould by a qualified surveyor who would be unaware of their health status. The housing stock in the catchment area of the hospital is varied and includes high rise flats, tenement buildings, council housing and modern houses built within the last five years. Each dwelling would be surveyed in a standard fashion as detailed below.

3.7.1 Temperature and relative humidity in dwelling

Spot temperature and relative humidity (RH) measurements were recorded outdoors and in the centre of each room in the dwelling using a *Whatman R 200* digital hygrometer. Temperature measurements were compared to a mercury in glass thermometer to ensure that the hygrometer had acclimatised when moving around within the property. As the recording of a mean indoor temperature and RH may obscure the presence of a cold damp room within the property, measurements were recorded within in each room in the dwelling. The minimum, maximum and mean indoor temperatures and the maximum and mean indoor relative humidities for each dwelling were calculated for statistical analysis.

As the kitchen is often a major source of moisture production, the temperature and RH in this room were used to calculate the dew-point temperature, that is the temperature at which the air would become saturated with water such that condensation would occur. This dew-point temperature was compared with the lowest temperature recorded within the dwelling in order to assess the likelihood of condensation occurring.

3.7.2 Quantifying damp in dwelling

Damp measurements were obtained using a commercially available electronic, conductance type moisture meter (*Protimeter Surveymaster*). Measurements were taken from three points just above skirting board height, on each wall (usually the middle and either end), in every room in the dwelling. Damp measurements at these sites are likely to detect both condensation and rising damp, but may miss damp secondary to other causes. Occasionally measurements were not obtained if the wall was tiled or access restricted.

At each point where a damp measurement was obtained, the severity was graded semi-quantitatively depending on the percentage scale deflection on the meter (table 7). Measurements were usually obtained from a total of 60 to 70 points in each dwelling. The highest grade of damp severity recorded (scale 0 - 4) and the sum of all the individual damp measurements (total damp score) were used as measures of damp severity in the statistical analysis.

Table 7. Grading of damp severity at point of measurement, calculated from the percentage scale deflection on damp meter

Severity Grade	Percentage scale deflection
0	<10%
1	10 - 25%
2	26 - 50
3	51 - 75%
4	>76%

3.7.3 Quantifying mould growth in dwelling

Mould growth within a dwelling is typically a result of condensation and as such the distribution of mould growth can give an indication of areas affected by condensation. These often include room corners of external walls where poor ventilation and low wall temperatures create optimal conditions for condensation. A mouldy area due to condensation may be found to be relatively dry when inspected with a moisture meter, if condensation has not occurred for several days. For these reasons it was felt important to attempt to quantify mould growth in the dwelling. The presence and severity of visible mould growth on each wall in each room of the dwelling was graded subjectively on a scale of 0 (absent), 1-trace, 2-obvious but localised and 3-obvious and widespread. The sum of these grades (total mould score) was used to quantify the amount of mould. A total mould score of three or greater was arbitrarily chosen to signify significant mould growth. Such a score could either represent one area of extensive mould or three areas of traces of mould.

3.8 Data analysis

3.8.1 Coding of data for analysis

The questionnaire and housing survey data were coded for computer analysis and transferred to a floppy disc in a tabulated format by a firm of independent data processors who were unaware of the purpose of the study. The data were then loaded into a statistical software package using a 486 personal computer. A random selection of the original questionnaire and survey results were compared with the data in the statistical package to ensure the data had been transferred accurately.

3.8.2 Conditional versus unconditional statistical analysis

Control subjects were frequency matched for gender and age to within five years in order to achieve a control group similar to the selected cases in terms of age and sex distribution. It was not anticipated that either of these variables would be associated with the exposure under study i.e. damp housing. In such circumstances, a statistical analysis that accounts for this matching is unnecessary and may lead to a less precise estimation of odds ratios (227-228). It was therefore decided that the matching of asthmatic and control subjects would be ignored, but a stratified analysis would also be performed where appropriate.

3.8.3 Calculation of odds ratios

Comparisons between categorical groups were made using the chi-square test with the odds ratio and its confidence interval stated where appropriate. The odds ratio can be regarded as the ratio of the odds of exposure in subjects with asthma relative to those without. An odds ratio greater than unity suggests a positive association between asthma and exposure under study, for example the presence of damp in the home. The confidence intervals for the odds ratio then give an estimate of the statistical significance of the association. If the 95% confidence intervals do not include unity then the association can be regarded as statistically significant at the 5% level.

3.8.4 Univariate statistical analysis

Continuous variables were compared using the students t-test or Wilcoxon Sum Rank

test depending on whether they were known to have a normal distribution. The total damp and total mould scores for surveyed dwellings were both positively skewed and their logarithmic transformations were used for statistical analysis. Linear associations between two continuous variables were assessed using the Pearson or Spearman correlation coefficients, depending on whether the variables were normally distributed.

3.8.5 Multivariate analysis to control for confounding variables

To control for potentially confounding variables more complex statistical methods were required, namely logistic regression and multiple linear regression analysis. If there was a binary outcome, for example when comparing the proportion of cases and controls exposed to damp in the home, multiple logistic regression models were constructed using the maximum likelihood method. Asthma would be entered as the response or dependent variable and the presence of damp and all possible confounding variables entered as explanatory covariants. Adjusted odds ratios and their 95% confidence intervals were then calculated from the logistic regression coefficients. When controlling for the effect of confounding on the correlation between two continuous variables, multiple linear regression models were constructed using the stepwise method and regression coefficients and their 95% confidence intervals quoted together with the associated p-value.

3.8.6 Statistical package

Analysis was performed using the Statistics Package for Social Sciences (SPSS for Windows, Release 6.0) (229). A significance level of 5% was used.

3.8.7 Power calculation

Assuming the proportion of controls exposed to damp in the home was 0.4 (97, 123, 146), it was estimated that a sample size of 90 asthmatic and 180 control subjects was required to detect an odds ratio of 2.0 at the 5% significance level with 80% power.

Chapter 4

Results

4.1 Demographic characteristics

Two hundred and ninety-eight (102 asthmatic and 196 control) subjects were entered in the study and completed a questionnaire. Their demographic characteristics are listed in table 8. One hundred and fourteen (38%) of the study group were male and the mean subject age was 26 years. There were 83 children under 16 years of age and for the majority (84%) of these the mother completed the questionnaire for the child. Asthmatic and control subjects were matched for age, gender and area of residence but several differences were identified between the two groups, particularly relating to employment status, household income and smoking status.

4.1.1 Employment status and household income

The employment status of all adults (age > 16 years) in the subject's household were recorded. Adult asthmatic subjects were less likely to be in full time employment than adult control subjects (35% Vs 55%, $X^2 = 10.5$, $p = 0.001$). Unemployment was slightly greater in the control group (12% Vs 5%) but adult asthmatic subjects were more frequently regarded as either temporarily or permanently sick or disabled (34% Vs 8%) and therefore unable to work.

The estimated net weekly household income was withheld by 15 (5%) respondents. Household incomes were generally low with a median of £160 per week (£8320 per annum). Net weekly incomes were lower in asthmatic compared with control subject households, with a greater proportion having a net income below £200 per week (75% Vs 60%, $X^2 = 5.95$, $p = 0.02$).

4.1.2 Housing tenure

There was a trend for more asthmatic subjects to live in rented as opposed to owner occupied dwellings (66% Vs 55%, $X^2 = 3.10$, $p = 0.08$). There were no differences in the proportion of asthmatic and control subjects who were renting, living in local authority (69%), co-operative (22%), Scottish Homes (7%), or privately rented (2%) accommodation. No differences in duration of tenancy were identified between asthmatic and control subjects (mean 7.4 years).

Table 8. A comparison of socio-demographic characteristics between asthmatic and control subjects.

Figures represent number (percentage) unless otherwise stated.
p-values are calculated from chi-square test or students t-test.

	Asthmatic (n=102)	Control (n=196)	p-value
Age (yrs), mean(SD)	26.4 (12)	25.8 (12)	ns
Sex, male	36 (35)	78 (40)	ns
Persons/room, mean (SD)	0.65 (0.23)	0.65 (0.23)	ns
Employment Status#			
Employed	36(35)	108(55)	p=0.001
Unemployed	5 (5)	23 (12)	
Sickness benefit	35 (34)	16 (8)	
Housewife	18 (18)	41 (21)	
Other	8 (8)	8 (4)	
Housing Tenure			
Rented	67 (66)	108 (55)	ns
Duration Tenancy (yrs), mean(SD)	7.4 (6.5)	7.4 (7.0)	ns
Net household income <£200 per week	76 (75)	118 (60)	p=0.02
Heating			
Central	67 (66)	121 (62)	ns
Gas fire	63 (62)	90 (46)	p=0.01
Paraffin	8 (8)	12 (6)	ns
Gas cooking	66 (65)	112 (57)	ns
Respondent Smoking Status			
Non-smoker	48 (47)	82 (42)	p=0.001
Ex-smoker	25 (24)	18 (9)	
Smoker	29 (29)	96 (49)	
Smoker in Household	60 (59)	137 (70)	p=0.05
Household pet	57 (56)	90 (46)	ns

employment status of adult respondent or parent of child.
(ns=not significant).

4.1.3 Smoking status

Control subjects were more likely to be current cigarette smokers (39% Vs 20%, $X^2=11.3$, $p=0.001$). There was a higher proportion of ex-smokers in the asthmatic group suggesting some patients had stopped smoking because of their asthma. The proportion of life long non-smokers in each group was similar. The proportion of households with no current cigarette smokers was slightly greater in the asthmatic than in the control group ($X^2=4.04$, $p=0.05$). The number of non-smoking subjects who lived with a smoker were similar in both groups ($X^2=1.71$, $p=0.2$).

4.1.4 Pet ownership

Pet ownership was common and tended to be higher in asthmatic (56%) compared with control (46%) subject households. This trend was greatest for dog ownership (38% asthmatic subject compared with 28% control subject households, $p=0.07$) whereas cat ownership was equally prevalent in both groups (15%). No differences were observed in the ownership of either smaller pet animals or birds.

4.1.5 Type of heating used in the dwelling

The different methods of heating used in the dwellings of asthmatic and control subjects were compared. Central heating was the commonest method used to heat the homes of asthmatic (66%) and control (62%) subjects. Gas fires were installed in 153 (51%) dwellings and were used more frequently in the homes of asthmatic compared with control subjects (62% Vs 46%, $p=0.01$). There were no differences in the proportion of dwellings with electric fires (28%) or paraffin heating (6.7%). Only two control subjects had coal fires.

4.1.6 Moisture production in dwelling

Moisture within a dwelling is generated by normal living activities and is proportional to the number of occupants. There was no difference in the mean number of occupants in the households of asthmatic or control subjects. The practise of washing and drying clothes indoors can be a major source of moisture production. The majority (95%) of the asthmatic and control subjects did their laundry at home.

Seventy percent of respondents admitted to drying the majority of their washing indoors, usually over radiators or clothes-horses in the kitchen. Few asthmatic subjects (10%) dried clothes outdoors, whereas 55 (28%) controls dried some of their clothes outdoors ($X^2=13.1$, $p=0.0003$).

Gas cooking produces up to a third more water vapour than other methods of cooking. The use of a gas stove was the commonest method of cooking in both the households of asthmatic (65%) and control (57%) subjects and did not differ significantly between the two groups ($X^2=1.59$, $p=0.2$).

4.2 Prevalence of respiratory symptoms

4.2.1 Asthmatic subjects

All 102 subjects with physician diagnosed asthma had respiratory symptoms such as wheeze, chest tightness and cough. Seventy-three (71%) patients had symptoms at least several times a week and 36 (35%) had symptoms most days or nights. The majority (88%) also experienced dyspnoea on exercise for example when walking uphill or climbing a flight of stairs. The mean (SD) age of onset of these respiratory symptoms was 12 (12) years and mean (SD) duration of symptoms 13 (11) years.

The asthma severity scores ranged from 2 to 24 with a mean (SD) of 11.3 (5.1). Only two patients did not use an inhaled bronchodilator, at least on an occasional basis and 92 (90%) were taking prophylactic therapy in the form of inhaled corticosteroids or sodium cromoglycate. The FEV_1 was greater than 80% of predicted normal in 57 (60%) of patients.

4.2.2 Control subjects

The prevalence of respiratory symptoms in control subjects was also assessed. Fifty-seven (29%) had symptoms of wheeze or chest tightness at sometime in the past, 33 (58%) of whom were cigarette smokers. Fifty-one (26%) of the controls had previously consulted their general practitioner for respiratory symptoms, but only 35 (18%) made a consultation within the previous 12 months. Twenty-three (12%) non-smoking control subjects had symptoms of wheeze and chest tightness on a

frequent basis, raising the possibility of them having asthma. As explained in section 3.1.2, a sub-analysis of the housing survey data was proposed excluding these 23 control subjects, to estimated if significant bias was introduced by including a small number of subjects with probable asthma in the control group.

4.3 Self reported damp and condensation

Respondents were questioned about the presence of damp and condensation within their home and requested to grade these subjectively as either a nuisance or a serious problem (table 9). Serious damp or condensation in the current dwelling was reported more frequently by asthmatic (33%) than control subjects (11%), (OR 4.1, 95% CI 2.3 to 7.6, p=0.0001).

Table 9. Prevalence and severity of self-reported damp and condensation.

Figures represent numbers (percentages).

	Asthmatic (n=102)	Control (n=196)	Total (n=298)
Damp			
None	67 (66)	150 (76)	217 (73)
Nuisance	9 (9)	28 (14)	37 (12)
Serious	26 (26)	18 (9)	44 (15)
Condensation			
None	55 (54)	130 (66)	185 (62)
Nuisance	26 (26)	49 (25)	75 (25)
Serious	21 (21)	17 (9)	38 (13)

There were significant trends for the prevalence of self-reported measures of damp to rise with increasing severity of asthma (table 10), such that those patients labelled as having severe asthma had the highest prevalence of any damp/condensation in the dwelling (X² trend=8.72, p=0.003) and more frequently regarded this as a serious problem (X² trend=30.5, p=0.00005).

Table 10. Prevalence of damp in dwellings of asthmatic and control subject.
 Figures represent numbers (percentages)

Self reported Damp measures	Controls (n=196)	Asthmatic Subjects *			X ² trend	p-value
		Mild (n=33)	Moderate (n=32)	Severe (n=32)		
Any damp or condensation in home	76 (39)	16 (48)	18 (56)	20 (62)	8.72	0.003
Serious damp or condensation in home	22 (11)	6 (18)	12 (37)	15 (47)	30.5	0.00005
Unable to use room because of damp	27 (14)	3 (9)	9 (28)	13 (41)	14.1	0.0002
Unable to use room because of cold	20 (10)	0 (0)	9 (28)	11 (34)	16.0	0.00006
Frequently unable to keep home warm	42 (21)	6 (18)	10 (31)	17 (53)	12.4	0.0004
Evidence of damp in previous house	56 (28)	12 (36)	16 (50)	17 (53)	11.3	0.0008
Moved because of damp in last house	20 (10)	5 (15)	8 (25)	8 (25)	8.33	0.004

* Severity score not available in 5 cases due to incomplete data

There were also trends for patients with more severe asthma to claim there were rooms in their home they were reluctant to use either because of damp (X^2 trend=14.1, $p=0.0002$) or cold (X^2 trend=16.0, $p=0.0001$) and admitted that living in such conditions caused financial, social and overcrowding problems. They were also frequently unable to heat their home adequately during winter months (X^2 trend=12.4, $p=0.0004$). When questioned about previous living conditions, subjects with severe asthma were more likely to report living in a previous home with evidence of damp or mould (X^2 trend=11.3, $p=0.001$) and to have moved from their previous dwelling because of damp (X^2 trend=8.3, $p=0.004$).

4.4 Independent survey of housing conditions

4.4.1 Comparison of surveyed and non-surveyed dwellings

Housing surveys were conducted for 75% of the sample. A comparison between surveyed ($n=222$) and non-surveyed ($n=76$) households showed no difference in socio-demographic characteristics including subject gender, age, household size, housing tenure, duration of tenancy, net weekly household income, cigarette smoking, pet ownership or self reported dampness. The only significant difference concerned employment, where fewer respondents in the surveyed households were employed ($X^2=7.47$, $p=0.01$). All subsequent analyses are based on the 222 (90 asthmatic and 132 control) subjects whose dwellings were surveyed.

4.4.2 Prevalence of damp and mould in dwellings

One hundred and two (46%) of the dwellings surveyed were categorised as dry. Dampness was detected using the moisture meter in 112 (51%) homes with 43 (19%) containing at least one area of grade 3 or 4 (severe) damp. Mould growth was observed in a total of 57 (26%) dwellings and in 33 (15%) homes was classified as significant (total mould score ≥ 3). The total damp scores ranged from 0 to 85 with a median of 6. Total mould scores were likewise positively skewed and their logarithmic transformations were used to normalise the distributions for statistical analysis. There was a significant correlation between a dwelling's total mould and total damp scores ($r=0.51$, $p=0.0005$) implying dwellings with extensive mould growth also tended to have areas of severe damp (figure 2). Only 8 (14%) dwellings had evidence of visible mould growth in the absence of damp detected by the moisture meter.

4.4.3 Comparison of self-reported and survey measures of damp

When comparing the results of the independent damp survey with measures of self-reported damp, there was a tendency for both asthmatic and control subjects to under-estimate damp. Damp was detected in the dwellings of 21 (52%) asthmatic and 27 (32%) control subjects who claimed their homes were dry. Fourteen subjects (3 asthmatic and 11 controls) who claimed there was no damp in the home had areas of mould growth observed by the surveyor. Agreement between self-reported damp and the findings of the surveyor occurred in 83 (63%) homes of control and 56 (63%) asthmatic subjects. This association between self-reported and surveyor confirmed damp was statistically significant ($X^2=15.6$, 0.00008), but the degree of correlation between the two measures was low ($r_s=0.26$).

4.4.4 Identification of possible confounding variables

Three housing condition groups based on the surveyors findings (dry, any damp, any mould) were compared on a background of possible confounding socio-economic variables. Two significant differences were identified relating to the presence of mould growth in the home. Those homes in which the respondent was unemployed had higher prevalences of mould than those in which the respondent was in paid employment (31% Vs 19%, $X^2=4.57$, $p=0.03$). Homes in which the net household income was under £200 per week had higher prevalences of mould than those in which the weekly income was over £200 (30% Vs 17%, $X^2=4.24$, $p=0.04$). No difference was observed in the prevalence of damp with either of these socio-economic variables but there was a trend for more damp to be found in rented than owner occupied dwellings (56% Vs 42%, $X^2=3.81$, $p=0.051$).

Homes with damp or mould did not differ from those classified as dry in either the number of children in the household, total number of household members, degree of overcrowding, subject's gender, subject's age band, respondents marital state, subject's smoking status or the presence of any smoker in the household.

Two housing characteristics associated with the presence of damp and mould growth in dwellings were identified. First, dwellings where gas was used for cooking were more likely to have an area of damp recorded (O.R. 2.14, 95%CI 1.23 to 3.72) than dwellings in which electric stoves were used. Second, dwellings with central heating

had significantly lower prevalences of both damp (O.R. 0.49, 95%CI 0.29 to 0.84) and mould growth (O.R. 0.37, 95%CI 0.2 to 0.68) than dwellings relying only on other forms of heating.

4.5 Housing conditions and asthma

4.5.1 Prevalence of damp in dwellings

One hundred and twelve (51%) of dwellings surveyed had some areas of damp. A comparison of dampness measures in the dwellings of asthmatic and control subjects is listed in table 11. Damp areas were detected in 58 (64%) homes of asthmatic subjects compared with 54 (41%) homes of control subjects (Odds Ratio 2.62 95%CI 1.50 to 4.55, $p=0.0006$). There was also a significant trend for a higher prevalence of any damp (X^2 trend=16.2, $p=0.00006$) and also for severe (grade 3 or 4) damp (X^2 trend=8.2, $p=0.004$) to be recorded in the dwellings of subjects with increasingly severe asthma.

In some asthmatic subjects the duration of tenancy at their current dwelling was less than the duration of their asthma. The prevalence of damp in dwellings of only those asthmatics who had developed symptoms whilst living at their current address ($n=39$) was compared with that of the control group. Damp was detected in 27 (68%) homes of this group of asthmatic subjects, which was again more prevalent than in the homes of the controls (O.R. 3.1, 95%CI 1.4 to 6.7, $p=0.003$).

4.5.2 Prevalence of mould in dwellings

Mould growth was found in 27 (30%) dwellings of asthmatic subjects and in 30 (23%) control dwellings. Again there was a trend for a higher prevalence of any mould (X^2 trend=4.24, $p=0.04$) and in particular significant mould growth (X^2 trend=8.1, $p=0.005$) in the dwellings of subjects with increasingly severe asthma.

Table 11. Prevalence of observed damp and mould in dwellings of asthmatic and control subjects.
 Figures represent numbers (percentages)

Damp measures	Controls (n=132)	Asthmatic Subjects*			X ² Trend	p-value
		Mild (n=29)	Moderate (n=29)	Severe (n=27)		
Observed damp						
Any damp	54 (41)	13 (46)	20 (69)	21 (78)	16.3	0.00005
Serious damp ¹	20 (15)	4 (14)	8 (28)	10 (37)	7.57	0.006
Observed mould						
Any mould	29 (22)	5 (17)	8 (28)	12 (44)	4.64	0.03
Significant mould ²	14 (11)	3 (10)	7 (24)	9 (33)	10.0	0.0015

* Severity score not available in 5 cases due to incomplete data.

1 Dwellings in which at least one damp measurement was of grade 3 severity.

2 Dwellings with total mould score greater than or equal to 3.

4.6 Location of damp in the dwelling

The location of the damp in the dwelling was recorded in 180 (112 control and 68 asthmatic) cases and is summarised in table 12. Damp areas were detected in the bathroom in 72%, kitchen in 56%, bedrooms in 54% and living rooms in 20% of dwellings categorised as damp. Asthmatic subjects had a higher prevalence of damp in all locations with the exception of the living room, where the prevalence of damp was equally low in asthmatic and control dwellings.

Table 12. Location of damp within the dwelling.

Figures represent numbers (percentages).

p-values calculated from chi-square test.

Location of damp	Control (n=112)	Asthmatic (n=68)	O.R. (95%CI)#	p-value
Bathroom	33 (29)	32 (47)	2.1 (1.1 to 3.9)	0.02
Kitchen	27 (24)	25 (37)	1.8 (0.9 to 3.5)	0.07
Bedroom	24 (21)	25 (37)	2.1 (1.1 to 4.1)	0.025
Living room	13 (12)	7 (10)	0.87 (0.3 to 2.3)	0.8

#Odds Ratio with 95% confidence intervals.

4.7 Controlling for confounding variables

Preliminary univariate analysis identified several possible confounding variables which could influence the observed association between damp housing and asthma. These included unemployment, low household income, smoking status, the lack of central heating or the use of gas fires to heat the dwelling, the use of gas stoves for cooking and the practise of drying all clothes indoors. To control simultaneously for the effects of these variables as well as age, gender and pet ownership, logistic regression models were constructed with asthma as the response or dependent variable and the measures of damp or mould and the confounding variables entered as covariates.

Table 13. Crude and adjusted odds ratios for asthmatic subjects living in dwellings with evidence of damp.

Damp measure	Crude O.R. (95% C.I.)	Adjusted O.R.* (95% C.I.)
Self reported	(n=298)	(n=283)
Any damp or condensation in home	1.92 (1.18 to 3.12)	1.93 (1.14 to 3.28)
Serious damp or condensation in home	4.13 (2.26 to 7.55)	5.45 (2.81 to 10.6)
Unable to use room because of damp	2.25 (1.24 - 4.10)	2.15 (1.13 - 4.11)
Unable to use room because of cold	2.28 (1.17 - 4.44)	2.25 (1.09 - 4.63)
Frequently unable to keep home warm	1.91 (1.12 - 3.26)	2.03 (1.12 - 3.69)
Evidence of damp in previous home	2.11 (1.29 to 3.47)	2.55 (1.49 to 4.37)
Moved because last home was damp	2.28 (1.17 to 4.44)	2.08 (1.02 to 4.24)
Observed	(n=222)	(n=211)
Any damp	2.62 (1.50 to 4.55)	3.03 (1.65 to 5.57)
Serious damp	2.14 (1.09 to 4.21)	2.36 (1.34 to 4.01)
Any mould	1.46 (0.80 to 2.67)	1.35 (0.79 to 2.28)
Serious mould	2.23 (1.07 to 4.63)	1.70 (0.78 to 3.71)

* Adjusted for subject age, sex, weekly household income (above/below £200), unemployment, respondent smoker, other smoker in household and pet ownership. 15 subjects did not disclose household income and were not included in the logistic regression models. Odds ratios and 95% confidence intervals calculated from logistic regression coefficients.

The crude and adjusted odds ratios for the various measures of self-reported and surveyor observed damp are listed in table 13. The adjusted odds ratios for the various measures of damp in the dwelling differ little from the crude ratios, suggesting the variables included in the logistic regression models had a small confounding effect. The adjusted odds ratios for the presence of mould in the dwelling were lower than the crude ratios with 95% confidence intervals that included unity.

4.7.1 Exclusion of control subjects with asthma like symptoms

The odds ratios reported above are based on the assumption that none of the control subjects had asthma. As control subjects were selected at random from the general population this is unlikely. To estimate the degree of bias this may have introduced, odds ratios for the prevalence of damp in dwellings of asthmatic subjects were re-calculated excluding 23 non-smoking controls who reported symptoms suggestive of asthma. The resultant odds ratios (table 14) differ little from those listed in table 13.

Table 14. Re-calculated odds ratios excluding control subjects with asthma like symptoms

Damp measure	Crude O.R. (95% C.I.)	Adjusted O.R.* (95% C.I.)
Observed damp	(n=202)	(n=193)
Any damp	2.70 (1.52 to 4.79)	2.93 (1.54 to 5.59)
Serious damp	2.01 (1.00 to 4.03)	1.95 (0.90 to 4.23)
Any mould	1.42 (0.76 to 2.66)	1.12 (0.56 to 2.03)
Serious mould	2.18 (1.01 to 4.67)	1.72 (0.75 to 3.94)

* Adjusted for subject age, sex, weekly household income (above/below £200), unemployment, respondent smoker, other smoker in household and pet ownership. 15 subjects did not disclose household income and were not included in the logistic regression models. Odds ratios and 95% confidence intervals calculated from logistic regression coefficients.

4.8 Temperature and relative humidity

The mean outdoor temperature and relative humidity at the time of the survey of the dwellings were 11°C and 68% respectively, whilst the mean indoor temperature and relative humidity of surveyed dwellings were 17.3°C and 57% respectively. Figure 3 shows the relationship between mean indoor temperature and relative humidity. There was a statistically significant negative correlation between these two parameters, with the mean indoor RH rising as indoor temperature fell ($r = -0.45$, $p = 0.0001$).

4.8.1 Indoor temperatures in homes of asthmatic and control subjects

The lowest, highest and mean indoor temperatures were noted in each dwelling. The lowest indoor temperature recorded was generally one degree centigrade below the mean indoor temperature for the dwelling (table 15). There was no significant difference in the highest indoor temperatures recorded in the homes of asthmatic or control subjects (18.5 Vs 18.8°C respectively). The mean indoor temperature in the homes of asthmatic subjects was lower than that in the homes of control subjects, mean difference -0.8°C (95%CI difference -1.5 to 0.0°C).

Table 15. Indoor temperature and humidity measurements in homes of asthmatic and control subjects

	Asthma	Control	mean diff.	95%CI	p-value
Mean temp (°C)	16.7	17.5	0.8	0.01 - 1.5	0.05
Lowest temp (°C)	15.6	16.5	0.9	0.1 - 1.7	0.03
Mean RH (%)	57	56	1.0	-3.9 - 1.8	0.5
Highest RH (%)	63	61	2.0	-4.8 - 1.5	0.3

4.8.2 Indoor temperatures and use of central heating

Homes without central heating were slightly colder than those where this form of heating had been used in the two weeks prior to survey (table 16), mean difference -1.2°C (95%CI difference -1.9 to -0.4°C). There was no significant difference in the use of this mode of heating in homes of asthmatic or control subjects ($p = 0.4$).

Figure 3 Correlation between indoor temperature and RH

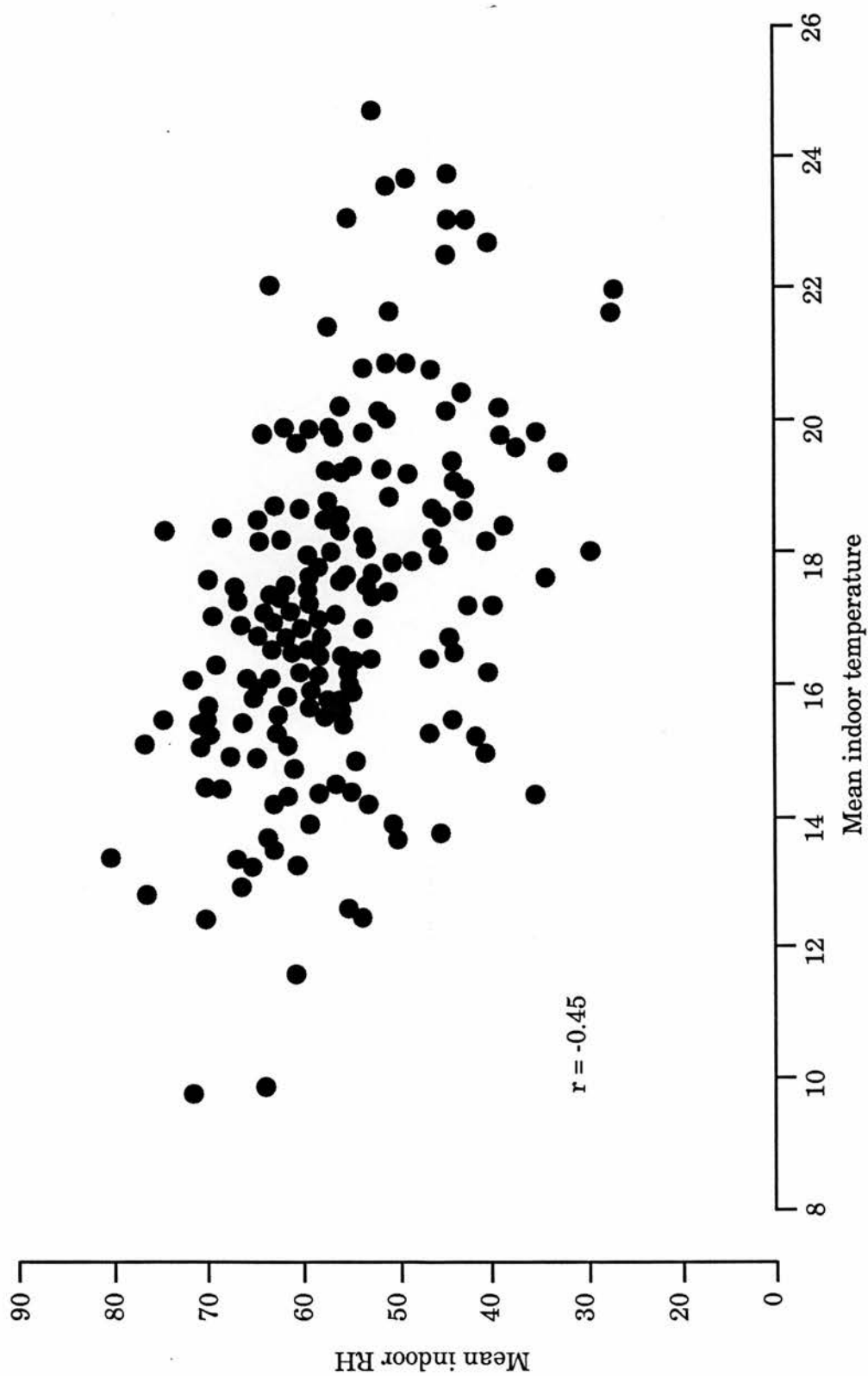


Table 16. Indoor temperature and humidity measurements in homes with and without central heating

	Centrally heated	No central heating	mean diff.	95%CI	p-value
Mean temp (°C)	17.7	16.5	1.2	0.4 - 1.9	0.002
Lowest temp (°C)	16.6	15.5	1.1	0.3 - 1.8	0.007
Mean RH (%)	55	59	4.0	2.0 - 7.0	0.002
Highest RH (%)	60	64	4.0	1.0 - 7.0	0.007

4.8.3 Indoor relative humidity in dwellings

No significant differences were observed in either the mean or highest indoor RH measurements recorded in the homes of asthmatic or control subjects (table 15). Dwellings without central heating had slightly higher mean RH levels (mean difference 4%, 95%CI 2 - 7, $p=0.002$), reflecting their lower indoor temperatures (table 16).

4.8.4 Indoor temperature and damp severity

There was a statistically significant negative correlation between damp severity and mean indoor temperature ($r= -0.40$, $p=0.0001$). Dwellings with evidence of damp had lower indoor temperatures (table 17) when compared with dry dwellings. Likewise, dwellings with evidence of mould were also colder, mean difference indoor temperature -1.6°C (95%CI -2.5 to -0.8°C , $p=0.0001$) than mould free dwellings.

4.8.5 Indoor humidity and damp severity

The relationship between indoor humidity and damp severity in dwellings is shown in figure 4. This shows a statistically significant positive correlation between damp severity and mean indoor RH ($r=0.39$, $p=0.0001$). Dwellings with evidence of damp or mould had higher levels of RH than dry homes (table 17 and 18). Problems with damp and mould occurred more frequently in dwellings with indoor relative humidities above 70%. Forty-five (23%) dwellings surveyed had at least one room in which the RH was greater than 70%. These dwellings had a higher prevalence of both damp and mould growth than those in which all RH measurements were below 70% (table 19).

Figure 4 Correlation between indoor RH and damp severity

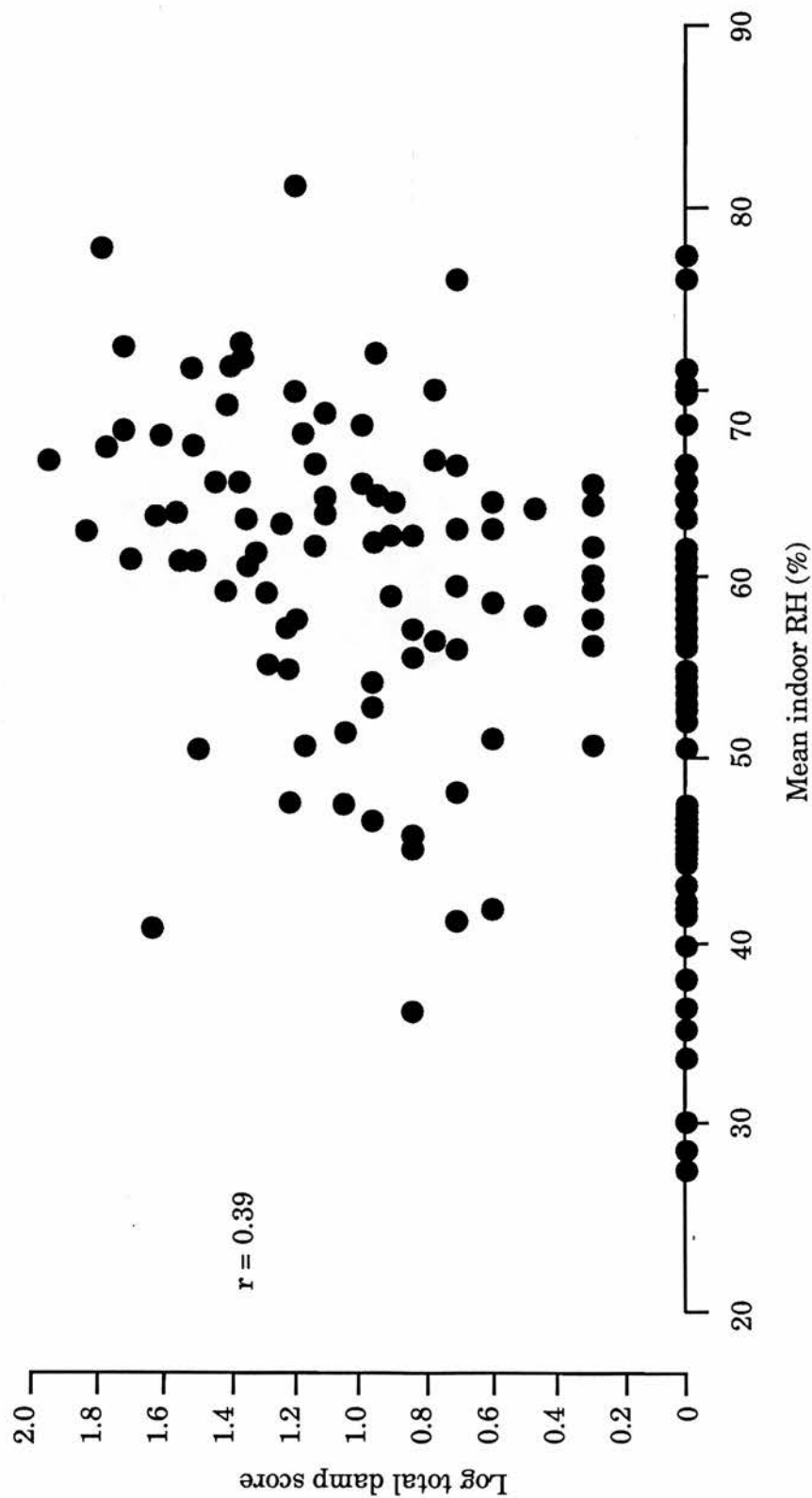


Table 17. Indoor temperature and humidity measurements in dry and damp dwellings

	Dry	Damp	mean diff.	95%CI	p-value
Mean temp (°C)	18.2	16.2	2.0	1.4 - 2.7	0.0001
Lowest temp (°C)	17.4	15.0	2.4	1.7 - 3.1	0.0001
Mean RH (%)	53	60	7.0	4 - 10	0.0005
Highest RH (%)	58	65	7.0	4 - 10	0.0005

Table 18. Indoor temperature and humidity measurements in mouldy and mould free dwellings

	No mould	Mould	mean diff.	95%CI	p-value
Mean temp (°C)	17.6	16	1.6	0.8 - 2.5	0.0001
Lowest temp (°C)	16.7	14.5	2.2	1.3 - 3.0	0.0001
Mean RH (%)	55	61	6.0	2 - 9	0.001
Highest RH (%)	60	66	6.0	2 - 9	0.001

Table 19. Prevalence of damp and mould in dwellings with high indoor RH.

Figures represent numbers (percentages)

Damp measure	RH < 70%	RH > 70%	Chi-square	p-value
Any damp	62 (41)	35 (78)	19	0.00001
Severe damp	19 (13)	15 (33)	10	0.001
Any mould	29 (19)	18 (40)	8.4	0.004
Severe mould	18 (12)	12 (27)	5.9	0.015

4.8.6 Dew point temperature and damp severity

The dew point temperature was calculated from standard psychometric charts using the temperature and RH recorded in the kitchen. The mean dew point temperatures were the same in asthmatic and control households at 8.5°C. The

dew point temperature was subtracted from the lowest indoor temperature recorded in each dwelling. The smaller the difference between these two temperatures, the more likely condensation would occur on colder wall surfaces.

The relationship between damp severity and the difference between the lowest indoor and dew point temperatures is illustrated in figure 5. This shows a statistically significant negative correlation with damp severity increasing as the difference between the temperatures approached zero ($r = -0.41$, $p = 0.001$). A similar, but slightly weaker correlation was identified with severity of mould in the dwelling ($r = -0.28$, $p = 0.001$).

4.9 Damp housing and asthma severity

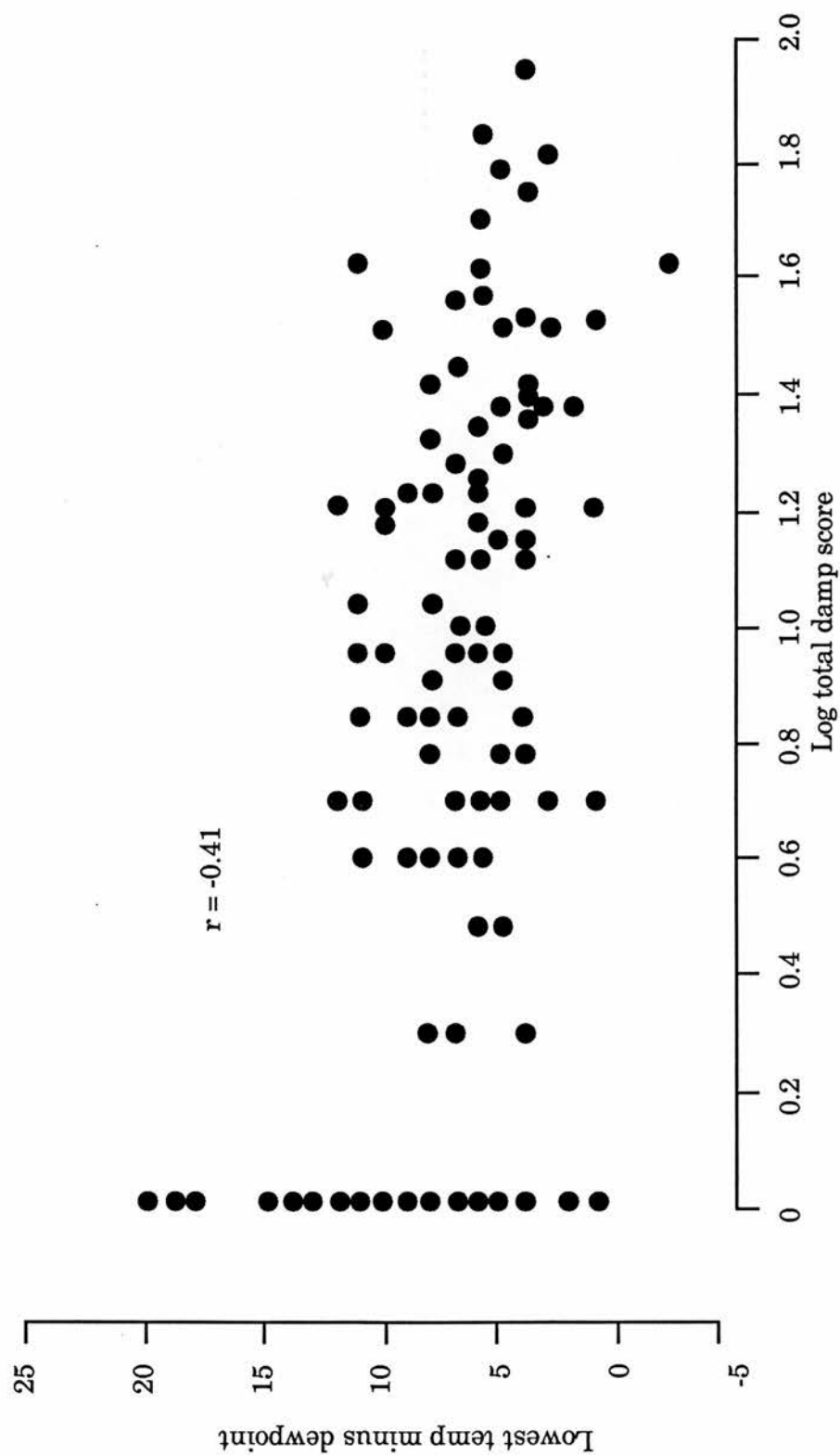
4.9.1 Comparison of surveyed and non-surveyed dwellings

Dampness surveys were conducted in 90 (88%) dwellings of asthmatic subjects. A comparison between surveyed and non-surveyed ($n = 12$) households revealed two significant differences. First, median duration of tenancy was longer in the surveyed group (6yrs) compared with the non-surveyed group (2yrs), ($p = 0.05$). Second, 22 (25%) of the surveyed group were employed compared with 7 (54%) of the non-surveyed group ($p = 0.05$). No other differences in socio-economic characteristics including age, sex, household size or tenure, weekly household income, cigarette smoking or self-reported dampness were identified.

4.9.2 Relationship between damp severity and asthma severity

Fifty-eight (64%) dwellings had evidence of damp and 27 (30%) visible mould growth. The relationship between asthma severity and quantity of damp in the home is shown in figure 6. This illustrates a statistically significant positive correlation between the asthma severity score and total damp score for the dwelling ($r = 0.30$, $p = 0.006$). A similar but slightly weaker correlation was observed between the asthma severity score and the total mould score for the dwelling ($r = 0.23$, $p = 0.035$). The greater the severity of damp or mould in the home, the more likely the patient was to have more severe asthma.

Figure 6 Correlation between damp severity and how close air temperature is to dewpoint



4.9.3 Controlling for confounding

Multiple regression analysis was used to control simultaneously for the influence of possible confounding factors such as household income, unemployment, respondent smoker, other smoker in house and pet ownership on the observed associations between asthma severity and severity of damp in the home. The adjusted correlation coefficients and their 95% confidence intervals are listed in table 20.

Table 20. Multiple regression analysis. Association between damp severity score and measures of asthma severity controlling for unemployment, household income, respondent smoking, other smoker in house and pet ownership.

Asthma severity measure	B (95% CI)	p-value
Asthma Severity Score	2.30 (0.53 to 4.07)	0.01
FEV ₁ (% predicted)	-11.9 (-19.4 to -4.39)	0.03
FEV ₁ /FVC ratio	-5.79 (-10.2 to -1.35)	0.01

B = regression coefficient

4.10 Damp housing and lung function

The relationship between lung function and severity of damp and mould in the home was assessed independently of other measures of asthma severity (figures 7a and 7b). Dampness severity in the dwelling was negatively correlated with the patient's FEV₁ percent ($r = -0.30$, $p = 0.006$) and FEV₁/FVC ratio ($r = -0.25$, $p = 0.023$). These observations remained statistically significant after controlling for both respondent smoking and the presence of a smoker in the household (table 20). Patients living in dwellings where the surveyor had confirmed evidence of damp had a lower FEV₁ percent (mean difference 10.6%, 95%CI 1.0 to 20.3) and FEV₁/FVC ratio (mean difference 5.4%, 95%CI -0.1 to 10.9) than patients living in dry dwellings. Overall, dampness severity within the dwelling accounted for approximately 9% of the variance in FEV₁.

Figure 7a Correlation between damp severity and FEV₁

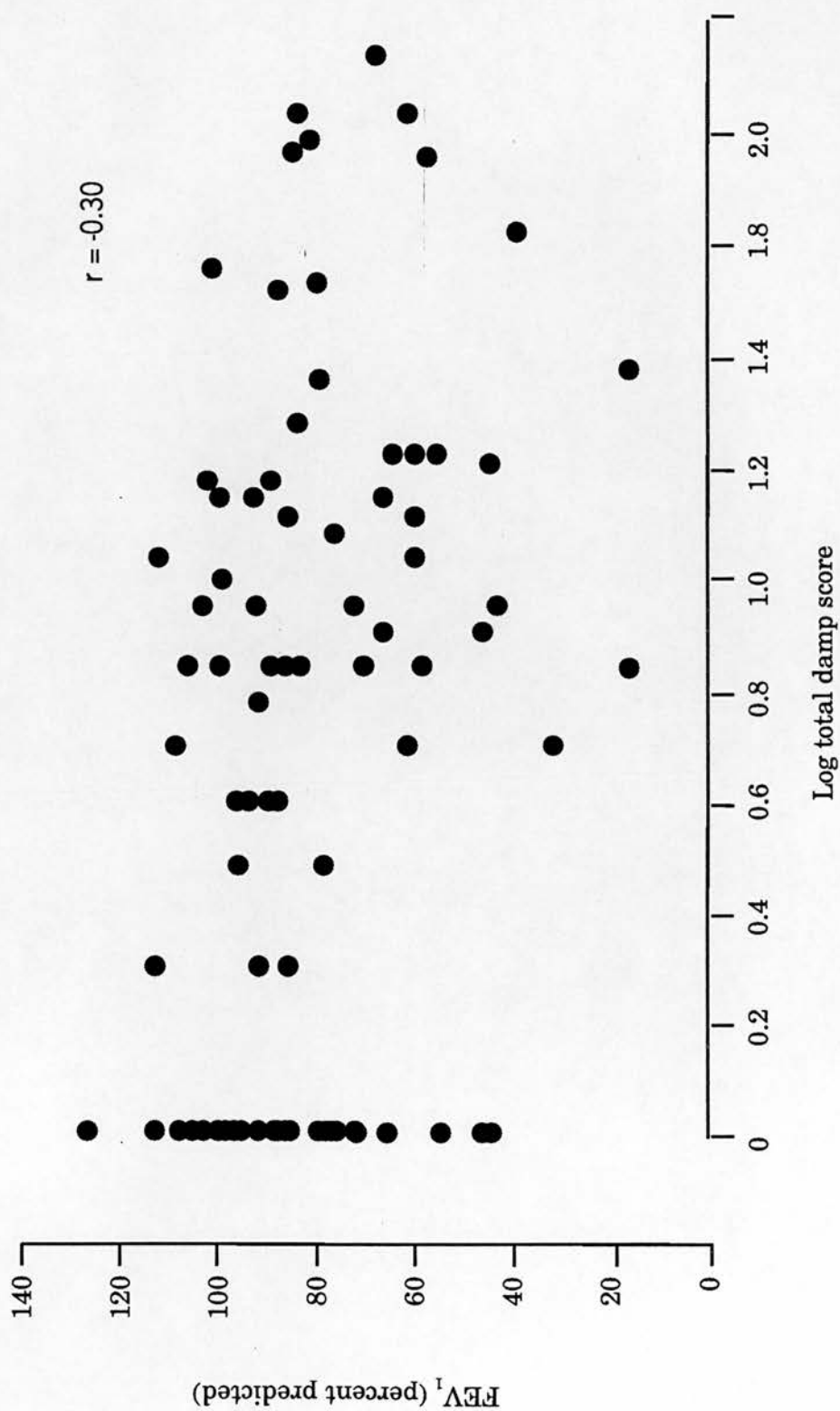
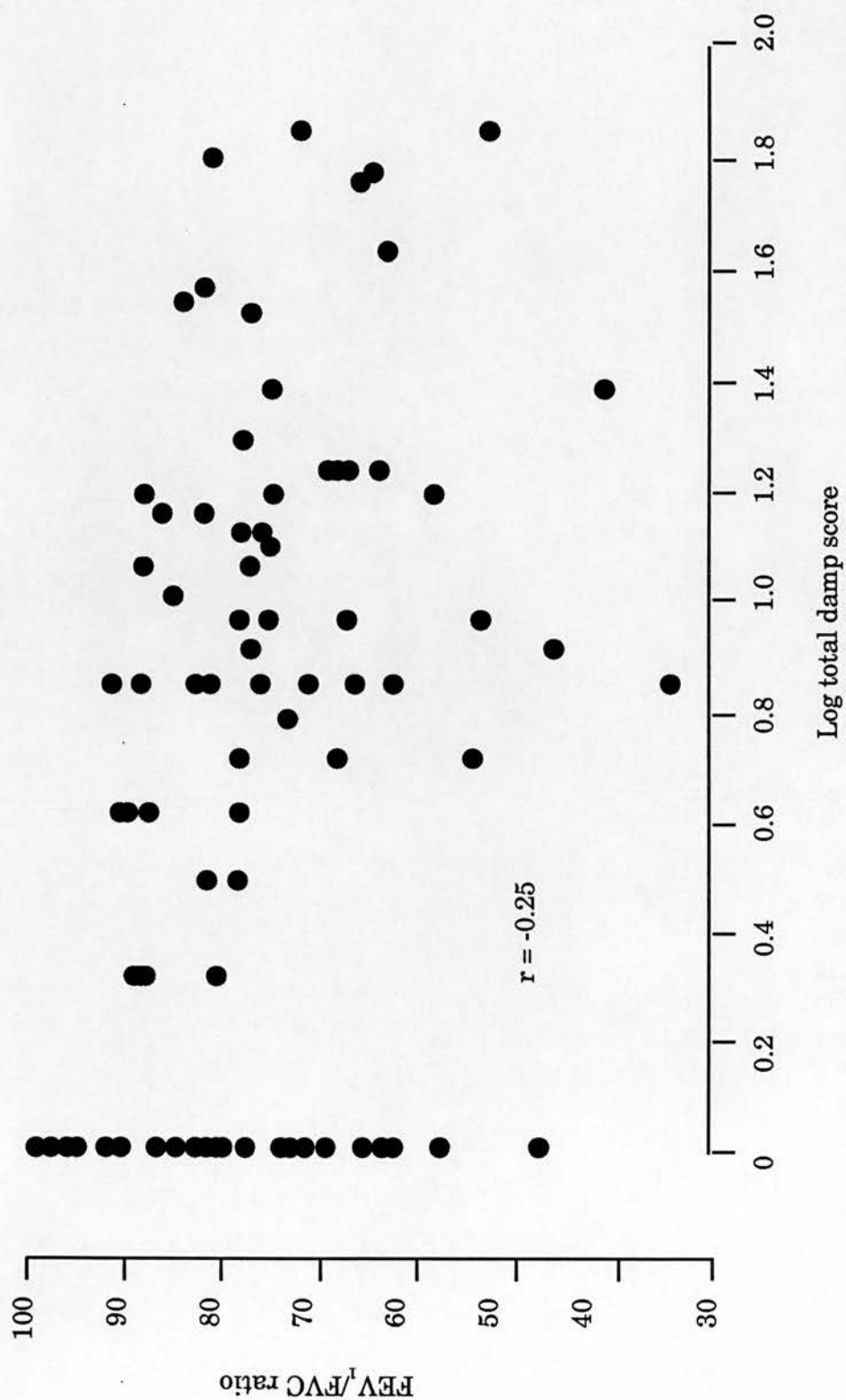


Figure 7b Correlation between damp severity and FEV₁/FVC ratio



4.11 Damp housing and asthma disability

The influences of damp and mould in the home on measures of functional asthma severity were also investigated. A weak statistical association was found between a patient's subjective health evaluation score and the total mould score for the dwelling ($r_s=0.21$, $p=0.04$). Seventy-nine (77%) of the asthmatics had spent time off work, school or stayed indoors because of their asthma in the twelve months prior to interview. Patients living in dwellings with evidence of damp tended to spend more time house-bound than those in dry homes (median 23 days Vs 11 days, $p=0.06$).

Thirty-two (43%) adults with asthma were registered as sick or disabled for employment purposes. When compared to other adult asthmatics, they were more likely to live in a home in which the surveyor had found evidence of damp, O.R. 4.44, (95%CI 1.37 to 14.4) or mould growth, O.R. 2.95 (95%CI 0.98 to 8.87). To control for possible confounding variables, logistic regression models were constructed with sick or disabled as the dependent variable and weekly household income, respondent smoker, other household smoker and the presence of any damp or mould entered as covariates. The adjusted odds ratios for the presence of damp or mould growth in the homes of adults registered sick were 6.79 (95%CI 1.67 to 27.6) and 1.40 (95%CI 0.37 to 5.29) respectively.

4.12 Damp housing, atopy and mould sensitivity

4.12.1 Skin prick allergy testing in asthmatic and control subjects

Ninety patients (88%) agreed to undergo skin prick allergy testing to common allergens and moulds whilst attending the asthma clinic. Following the completion of the damp surveys, letters were sent to control subjects inviting them to attend the hospital clinic for a clinical assessment to include skin prick allergy testing and lung function testing. Eighty-eight (45%) controls consented to allergy testing. A comparison between skin tested and non-skin tested control subjects showed no difference in subject sex, age, pet ownership, prevalence of respiratory symptoms or housing characteristics such as self reported or surveyor confirmed damp or mould. Control subjects who did attend were however more likely to be employed ($X^2=4.70$, $p=0.03$) and non-smokers ($X^2= 5.73$, $p=0.02$).

4.12.2 Prevalence of atopy

The results of the skin prick testing are shown in table 21. Seventy-seven (86%) asthmatic and 44 (50%) control subjects were atopic with one or more positive reactions to either house dust mite, cat or dog dander or pollens. There were no significant differences in the prevalence of atopy with sex or age, although there was a trend for atopy to be slightly less common in subjects over 40 years.

Table 21. Skin prick allergy testing to common allergens and indoor moulds.

Figures represent numbers (percentages).

	Asthmatic (n=90)	Control (n=88)
Common Allergens		
House dust	58 (64)	26 (29)
House dust mite	63 (70)	28 (32)
Cat dander	46 (51)	15 (17)
Dog dander	26 (29)	5 (6)
Pollens	46 (51)	22 (25)
ATOPIC (any of above)	77 (86)	44 (50)
Common Moulds		
Cladosporium	4 (4)	2 (2)
Alternaria	2 (2)	0 (0)
Penicillium	3 (3)	0 (0)
Aspergillus fumigatus	3 (3)	2 (2)
Aspergillus niger	4 (4)	0 (0)
Fusarium	3 (3)	1 (1)
Rhizopus	3 (3)	0 (0)
Sporobolomyces	1 (1)	0 (0)
Dry rot	1 (1)	0 (0)
Mould sensitive	15 (17)	5 (6)

4.12.3 Relationship between atopy and damp housing

The prevalences of atopy and house dust mite sensitivity in asthmatic and control subjects were compared with current and previous housing characteristics. Patients

who claimed to have lived in a previous home with evidence of damp were less likely to have a positive skin test reaction to house dust mite allergen than those who had lived in dry homes (67% Vs 87%, $X^2=5.62$, $p=0.02$). Patients living in homes in which the surveyor found evidence of damp were also less likely to have a positive reaction to HDM allergen than those living in homes confirmed to be dry (69% Vs 96%, $X^2=8.04$, $p=0.005$).

Control subjects living in homes with self-reported evidence of damp were more likely to be HDM allergen sensitive than those claiming their homes were dry (57% Vs 28%, $X^2=6.76$, $p=0.01$), but this observation did not hold for surveyor confirmed damp where the prevalence of HDM sensitivity was equal in control subjects living in dry (40%) and damp (39%) homes. There were no statistically significant associations between atopy and housing characteristics.

4.12.4 Mould sensitivity and housing characteristics

Fifteen (17%) asthmatic and 5 (6%) control subjects were classified as mould sensitive with at least one positive reaction to the nine moulds tested. Only one asthmatic subject had a positive reaction to a mould but was classified as non-atopic on the basis of the other skin tests. The prevalence of mould sensitivity in patients was similar in those living in dry (18%), damp (15%) and mouldy (25%) dwellings. Only those asthmatics living in homes classified as having significant mould (total mould score for dwelling ≥ 3) were more likely to have a positive skin test to one of the nine moulds tested (35% Vs 11%, Fisher's exact test, two-tailed $p=0.03$). Mould sensitivity in control subjects was too low to compare meaningfully with their housing characteristics.

4.13 Summary of study results

1. Reasonable agreement was found between the respondent's reporting of damp and condensation in the home and the presence of damp detected by use of a moisture meter during the survey. There was a tendency for both asthmatic and control subjects to under-estimate the presence of damp in the home, even in the presence of mould growth.

2. Dwellings in which the surveyor found evidence of damp or mould were significantly colder with correspondingly higher relative humidities. Indeed there was a significant negative correlation between mean indoor temperature and severity of damp in the home. Colder homes are more prone to damp and condensation.
3. There was a good correlation between the severity of damp and the extent of mould growth observed in a dwelling.
4. Asthmatic patients attending a hospital asthma clinic were two to three times more likely to live in a dwelling with evidence of either self-reported or confirmed damp, than were a group of randomly selected age and sex matched controls from the general population, living in the same area of Glasgow. These associations persisted after controlling for potential confounding variables.
5. There were significant trends for the prevalence of both self-reported and surveyor observed damp to rise with increasing severity of asthma. Furthermore, there were significant correlations between asthma severity and severity of airflow obstruction with the severity of damp and mould in the dwelling, suggesting a dose-response relationship.
6. There was no association between a subject's atopic status or sensitivity to the house dust mite and the presence of damp or mould growth in the home.
7. Asthmatic subjects living in dwellings with more extensive mould growth were more likely to have a positive skin prick allergy test to one of nine common indoor moulds.

Chapter 5

Discussion

5.1 Overview of study findings

This study confirmed an association between asthma and damp housing. Patients attending a hospital asthma clinic were two to three times more likely to have damp within their home than a group of age and sex matched control subjects. This association was strongest in those with moderate to severe asthma. To my knowledge, it is also the first study to identify a dose-response relationship between asthma severity and severity of damp in the dwelling. In particular the degree of airflow obstruction in asthmatic subjects was found to be significantly associated with severity of damp in the home.

Attempts to identify risk factors for a disease often have to rely on results of observational studies such as that described here. Such studies are particularly prone to several areas of criticism (230). Firstly, their design has to overcome areas of potential bias including investigator, non-response and respondent or recall bias. Secondly, the possibility of confounding variables must be considered and finally when these issues have been addressed, estimates can be made as to whether the observed association could have occurred by chance alone. It is important that these issues be discussed with relevance to the findings of this case-control study.

5.2 Potential bias in study design

5.2.1 Selection bias

The interpretation of results from a case-control study is often dependent on the suitability of the control group. Ideally, controls and cases should constitute populations identical in all respects except for the disease studied. It could be argued that the most appropriate control group for this study would have been to select age and sex matched non-asthmatic patients from another hospital out-patient department.

Asthmatic subjects aged between 5 and 45 years of age were selected for study. This age range was chosen to exclude other respiratory disorders with symptoms common to those found in asthma. The diagnosis of asthma in children under 5 years is problematic as they may develop temporary symptoms of wheeze and cough, especially following viral upper respiratory tract infections. Such symptoms are thought to be secondary to the relatively small diameter of their bronchi rather than to underlying asthma. The upper age limit of 45 years was imposed in an attempt to exclude subjects with smoking related airflow obstruction.

Due to the age range of the cases selected for study, there were practical difficulties in identifying a comparable population of patients attending another clinic within the hospital. It was also felt that control subjects selected from another hospital out-patient clinic might have represented a different sub-group of the general population. Any observed differences between the asthmatic and such a group of control subjects, could have had more to do with the epidemiology of the disease of the control subjects than with the disease study.

The control group in this study was randomly selected from the general population living within the catchment area of the hospital. The demographic characteristics of this control group were similar to those subjects recruited from Strathclyde in the Scottish house condition survey in 1991 (123). Both groups had similar levels of unemployment and long term sickness. The percentage of owner occupied dwellings was also similar in both groups. It is likely that the control subjects in this study were fairly representative of the general population living within the catchment area of the hospital.

As the control subjects were selected at random from the general population, a proportion of them would have respiratory symptoms consistent with a diagnosis of asthma. The prevalence of asthma in the general population in Britain is estimated at approximately 10%. In a random selection of 200 subjects, one would therefore expect up to 20 cases of asthma. The definition used to select cases for inclusion in the study was that physician diagnosed asthma. It was impractical to arrange for a respiratory physician to examine all control subjects to identify those with possible asthma. However, none of the control subjects had physician diagnosed asthma or had previously attended a hospital asthma clinic.

From questionnaire responses, twenty-three non-smoking control subjects had persistent symptoms of wheeze and chest tightness consistent with a diagnosis of asthma. These subjects were retained in the control group for the purposes of the statistical analysis as it was anticipated that their small number would be unlikely to significantly influence the results of the study. Any bias introduced by their inclusion would result in an under rather than over-estimation of the odds ratio for the prevalence of damp in dwellings of asthmatic subjects. A sub-analysis of the data excluding these 23 control subject had little influence on the reported odds ratios.

The selection of subjects with physician diagnosed asthma from a hospital out-patient department also introduces an element of selection bias. These subjects

are clearly not representative of asthma in the general population, but represent those at the upper end of the disease severity spectrum. The results of this study cannot therefore be extrapolated to all subjects with asthma. Indeed, there was an increasing prevalence of damp in the home with increasing severity of asthma. Those asthmatic subjects classified as having mild asthma had only a slight increase in prevalence of damp in the home compared with the control subjects. The results suggest that damp in the home may aggravate asthma severity and therefore be a risk factor for referral to a hospital asthma clinic.

5.2.2 Respondent and recall bias

Previous cross-sectional epidemiological surveys investigating the effects of damp housing on health have tended to rely on the same questionnaire to elicit information on both the subjects health and indicators of damp in the dwelling, giving the potential for the results to be influenced by respondent and recall bias (132, 231). Adult asthmatic subjects or the parents of asthmatic children may be more aware of condensation, damp or mould growth in the dwelling and the possible adverse effects they may have on health. They would be more likely to respond positively to questions related to the presence of damp in the dwelling than would healthy controls. In addition it could be argued that asthmatic subjects attending a hospital out-patient clinic would give more consideration to questions than control subjects selected at random from the general population.

The prevalence of self-reported damp and condensation in the homes of control subjects was slightly greater than that reported in the 1991 Scottish homes survey (39% Vs 27%), although the number of households with severe damp was similar (11% Vs 13.3%). The prevalence of self-reported damp and condensation in the dwellings of asthmatic subjects was much higher at 56% and was regarded as serious in 37% dwellings.

As a consequence of the increased prevalence of damp and condensation, asthmatic subjects were more likely to claim that there were rooms in their dwelling they were either reluctant or unable to use. This increased reluctance to use certain rooms caused more problems with overcrowding and significant financial, social and emotional problems for household members. This increased stress and disharmony within the home may have either adversely influenced a subject's asthma severity or their perception of symptoms.

It has been previously argued that such questionnaire responses could be influenced by respondent bias. To minimise such bias, all subjects were unaware of the purpose of the study at the time of interview and the questionnaire was constructed so that subjects were asked questions with regards to their health status before any questions regarding housing characteristics.

5.2.3 Respondent Vs surveyor reported damp

Previous studies comparing the results of dampness surveys conducted by an independent surveyor with the occupant's perception of damp in the dwelling have often found marked discrepancies (145-146). The Scottish homes study found that whilst there was reasonable agreement between the presence of self-reported damp and condensation and the presence of damp and condensation recorded by the surveyor, there were often quite marked discrepancies when assessing the severity of the problem.

In this study, the surveyor found evidence of damp in 41% dwellings of control subjects and in 15% dwellings this was graded as severe. These figures are similar to those recorded for self-reported damp. On further analysis of the results there were several discrepancies. The surveyor found evidence of damp in about a third of homes which would have been classified as dry from questionnaire responses. Overall, the agreement between the presence of self and surveyor-reported damp was only 63%. The agreement between the assessment of severity of any damp in the home is likely to have been lower.

This study confirms the advantages of obtaining independent survey reports of dwellings in health surveys. By adopting an independent, standardised, semi-quantitative approach to assessment of damp in each dwelling, the inaccuracies which arise when relying solely on self-reported measures of damp were avoided.

In order to assess respondent bias, the prevalence of self-reported damp in the dwellings of asthmatic and control subjects were compared to the findings of the independent surveyor. Both groups of subjects tended to under-report damp in their homes, sometimes even in the presence of visible mould growth. This suggests the asthmatic subjects were no more aware of the presence of damp in their home than were controls. Respondent bias was considered unlikely to have significantly influenced the association between asthma and self-reported damp.

Brunekreef came to a similar conclusion when he attempted to assess whether parents living in mouldy homes were more likely to report respiratory symptoms in their children than parents with dry homes (231). He found the relationship between respiratory symptoms and measures of lung function were similar in children living in mouldy homes and dry homes, suggesting parents in damp homes were not over-reporting respiratory symptoms in their children. Likewise, Verhoeff *et al* found a reasonable agreement between reported signs of damp in the home over the past two years and observed signs of dampness, after adjusting for mould removal (100). There was no evidence of over-reporting of damp or mould by the parents of children with respiratory symptoms relative to the parents of controls.

In contrast, Strachan concluded that his observation of an association between respiratory symptoms in childhood and mould growth in the home was likely to be due to respondent bias, with the parents of children with symptoms over-reporting damp in the home (131-132). This conclusion was based on finding no association between damp in the dwelling and either the number of general practitioner consultations for respiratory illnesses made by the child or demonstrable exercise induced broncho-constriction.

Strachan may have been too critical in the interpretation of his study findings. There are many factors which may influence whether a parent takes a child to see their General Practitioner. Furthermore, it is widely accepted that asthma in children is often not recognised by their parents and is often under-diagnosed in the primary care setting. Collection of retrospective attendance data in this way may not be particularly helpful.

Strachan's subjects were selected from the general population and the majority of children with asthma were likely to have had mild disease. Exercise induced broncho-constriction is seldom found in children with mild asthma (232) and the number of children in Strachan's study with significantly increased airway responsiveness was small, preventing meaningful comparisons with housing characteristics.

5.2.4 Investigator bias

Case-control studies cannot be conducted in the randomised, double blind, controlled manner now regarded as standard in studies of experimental design. They are therefore more prone to investigator bias. In this study, the questionnaire and

dampness survey data were collected in a blind fashion by independent researchers and then coded for computer analysis by individuals unaware of the purpose of the study. As dampness in the dwelling was measured objectively and the diagnosis of asthma had been previously established by an independent respiratory physician, investigator bias was effectively eliminated.

5.2.5 Non-response bias

By using an interviewer to administer the questionnaire, a high standard of data collection was maintained for all subjects and minimised non-response bias, which is a frequent problem in postal surveys. Although only 74% of subjects agreed to have their home surveyed, there was no significant differences in socio-demographic data or self-reported damp, as assessed by questionnaire responses, between those subjects whose dwelling had been surveyed and those where a survey had not been possible.

The subjects whose dwellings were surveyed were more likely to be unemployed, but this may reflect easier access to the dwelling by the surveyor during normal working hours. Similar observations were noted by Martin *et al* (145) who also used a surveyor to obtain objective measurements of damp in dwellings. It is unlikely that the non-surveyed dwellings differed significantly in terms of damp housing characteristics from those surveyed.

5.3 Confounding

As controls selected from the general population may differ significantly from patients attending a hospital out-patient clinic, it is important to try to identify any important differences between cases and controls and if possible control for these potential confounding variables in the statistical analysis of the study results.

5.3.1 Age, gender and area of residence

Asthmatic and control subjects were matched for gender, age to within five years and area of residence in Glasgow restricted to area post codes G51, G52 and G53. No significant associations were identified between gender, age and housing

characteristics. A higher proportion of dwellings in postal code area G53 were classified as damp, but there was no difference in the proportion of asthmatic or control subjects recruited from this area of the city.

5.3.2 Unemployment, long term sickness and household income

Where the head of the household is unemployed or suffers a long term sickness, it is likely they will have lower household incomes and may therefore live in poorer quality accommodation. The Scottish Homes survey found a higher prevalence of damp in the homes of such subjects, 20.2% dwellings compared with the mean of 11.7% dwellings in the Strathclyde region. This observation is of direct relevance to this study.

The asthmatic subjects in this study were more likely to be unemployed or regarded as sick or disabled because of their asthma and have lower net household incomes than controls. This could result in their living in poorer quality housing, which they would be less able to afford to heat adequately and therefore be more prone to damp. However, the association between asthma and the presence of damp in the home remained statistically significant when controlling for an unemployed respondent, the lack of anyone in the household in paid employment and a low net household income (above or below £200 per week).

The majority of asthmatic subjects lived in local authority housing and it is unlikely they were systematically allocated damp or mouldy dwellings. There was also little difference in the availability or use of central heating in the homes of asthmatic and control subjects, although the mean indoor temperature in the dwellings of asthmatic subjects was one degree centigrade lower than in the dwellings of control subjects. These results argue in favour of the observed relationship between damp housing and asthma being largely independent of household income.

5.3.3 Social class

Several studies have identified a higher prevalence of severe asthma and resulting morbidity and mortality in lower social classes (168-169). Various explanations have been proposed to account for these observations. Severe asthma may mean that individuals move down the social scale, but evidence for this is lacking, although chronic illness may limit employment choices and opportunities (160). Inadequate

treatment of symptoms or inability to afford allergen avoidance measures should also be considered (233). Strachan found that diagnostic labelling and drug treatment of wheezy children did not differ substantially by socio-economic status (170). The more likely explanation is that these individuals have more severe asthma as a result of greater exposure to exacerbating factors such as cigarette smoking and poor housing conditions.

5.3.4 Cigarette smoking

Cigarette smoking was less common in asthmatic subjects. It was anticipated that smoking would be associated with unemployment, lower social class and therefore indirectly with damp housing. Unlike the previous confounding variables, failure to control for the confounding effect of smoking may have led to an under-estimate of the odds ratio for damp in dwellings of asthmatic subjects. Smoking status was therefore retained as a significant variable in all the multi-variate statistical models.

The exposure of children to environmental tobacco smoke is known to have an adverse effect on their asthma. The failure to control for parental smoking in the homes of asthmatic children may have significantly influenced the results. The presence of any smoker in the home was therefore also controlled for in the analysis. It was accepted that this would give only a crude measurement of exposure to environmental tobacco smoke. Formal quantification of this exposure, for example by measuring urinary cotinine levels was not undertaken.

The results showed that both cigarette smoking by the subject and exposure to environmental tobacco smoke by a non-smoking subject had little influence on the observed association between asthma and the presence of damp in the dwelling. The relationship between severity of damp in the dwelling and the severity of airflow obstruction measured on spirometry was also independent of smoking. Cigarette smoking was considered not to have had a significant confounding influence.

5.3.5 Pet ownership

Despite the possible adverse effects of pet ownership on asthma severity, the households of asthmatic subjects were more likely to have a pet, often a dog. The effects of pet ownership on the presence of damp in the home are unknown, but

they are known to adversely influence asthma severity, including medication requirements. Pet ownership could therefore have adversely influenced the asthma severity scores. The associations between damp severity and asthma severity including degree of airflow obstruction remained statistically significant after controlling for pet ownership.

5.4 Why are dwellings of asthmatic subjects more prone to damp?

This study has confirmed the apparent relationship between damp housing and asthma. It was designed with the intent of reducing bias to a minimum and also with a view to controlling for important identified confounding factors. Accepting this is a true association, some consideration is required as to the possible explanations as to the cause of the excess damp in the dwellings of the asthmatic subjects. Although it was beyond the remit of the surveyor to comment on the cause of any damp found during the survey of a dwelling, this will be discussed in the next section.

5.4.1 Increased moisture production?

Increased moisture production within a dwelling will raise the relative humidity, increasing the likelihood of condensation formation on cold surfaces. Normal living activities generate moisture and will therefore be dependent to some extent to the number of occupants in the dwelling. We observed no significant differences in the number of occupants in the dwellings of asthmatic and control subjects. We did not however, enquire about the number of hours spent indoors by the occupants of the dwelling. As unemployment was more common in the asthmatic group, it is possible these subjects spent more time indoors and therefore generated more moisture than control subjects.

Moisture may be added by the use of gas stoves for cooking and un-flued gas or paraffin heaters, but most is generated by boiling water, bathing and washing and/or drying clothes indoors. The use of gas cookers and paraffin heaters was similar in the asthmatic and control groups, but asthmatic subjects used gas fires more frequently to heat their homes. This however, did not appear to influence the prevalence of damp in the home. The practise of drying clothes indoors was equally

common among asthmatic and control subjects, although the latter claimed to dry some clothes outdoors more frequently than did asthmatic subjects. Overall, there was little evidence to support a hypothesis that there was significantly greater moisture production in the homes of asthmatic subjects.

5.4.2 Physical properties of the dwelling?

As no discernible differences were observed in the amount of moisture produced in the dwellings of asthmatic and control subjects, the observed increased prevalence of damp in the homes of asthmatic subjects is likely to be due to differences in the physical properties of the dwellings such as state of disrepair and degree of insulation which influence indoor temperature, ventilation and penetration of moisture from outdoors.

Dwellings of asthmatic subjects were marginally colder than those of control subjects. Although the lowest indoor temperature recorded was always greater than the calculated dew-point temperature, external wall temperatures are likely to be several degrees cooler than those recorded in the centre of a room. The measurement of temperature and humidity at the wall surface would have been a better predictor of the risk of condensation.

There was no significant difference in the use of central heating in the dwellings of asthmatic or control subjects to account for the observed small difference in mean temperatures. This may suggest that the dwellings of asthmatic subjects were either more difficult to heat or were more prone to lose heat perhaps because of poorer insulation. Further information would have been required to assess these possibilities in more detail. Indoor temperature recordings were recorded at only one point in time in each dwelling. Continuous recordings of temperature and humidity using thermo-hydrographs to give seven day minimum, maximum and mean values would have been more desirable, but was outside the scope of this study.

Reduced ventilation in dwellings also predisposes to the development of condensation by maintaining higher indoor relative humidities. This is one of the more important predisposing causes of damp and in particular condensation in modern housing. In attempting to become more energy efficient, houses are being built more air-tight with draughts prevented, windows double glazed and chimneys

either absent or blocked. Moisture produced within the dwelling is therefore unable to escape and often allowed to disperse through the building into colder spaces such as unheated bedrooms, where condensation will occur on cold surfaces. It was not possible in this study to measure air exchange rates within dwellings and I was unable to determine if ventilation was significantly lower in the dwellings of asthmatic subjects.

The combination of low temperatures and reduced ventilation in dwellings creates ideal conditions for condensation. It follows that better heating and increased ventilation are required to tackle these problems. Such an ideal is unlikely to be practical in a climate such as that found in Britain. Many of the energy conserving measures introduced to conserve heat within dwellings and hence help keep fuel bills lower, also reduce ventilation. Any attempts to increase the ventilation of a dwelling during a cold British winter will also increase heating requirements and fuel bills, unless such a system incorporated some form of heat exchange mechanism.

In summary, although this study was not designed to identify the cause of damp found in surveyed dwellings, only minor differences in sources of potential moisture production within dwellings were identified. These were considered unlikely to account for the excess prevalence of damp in the dwellings of asthmatic subjects. It is more likely that the observed differences in damp prevalence between the two groups were related to differences in the physical properties of the dwellings, for example the state of disrepair and use of heat conservation measures such as double glazing and other forms of insulation.

These findings have important implications on the potential advice which could be offered to asthmatic subjects. It is unlikely that changes in personal lifestyle will significantly influence the severity of the damp problem in their dwelling. It is more likely that expensive alterations to the property would be required, which as previously stated, they are less likely to be able to afford.

5.5 Causality

Questions concerning the causes of diseases are often addressed by observational studies. The major disadvantage of such a study is their accepted inability to prove causation. Although this study identified an association between asthma and damp housing conditions, it is not possible to say whether an adverse change in housing conditions would influence either the prevalence or severity of asthma.

The problems of inferring causality from observational associations were addressed by Bradford Hill (230). Although he did not intend his paper to be used as a checklist of criteria for causality, I shall discuss with relevance to this study, the aspects of an observed association that he felt should be given special consideration before deciding the most likely interpretation of an association to be a causal relationship.

5.5.1 Statistical significance

Tests of statistical significance are frequently applied to the results of observational studies. Such tests play little part in deciding whether a relationship is causal. Statistical tests will give information as to the probability of the study results occurring by chance alone. They give little indication as to whether the observed association is real or spurious. If however the probability that the results occurred by chance alone is very small, then this might support the case for a causal association.

The crude odds ratio, calculated from the results of this study, for any damp to be present in the homes of asthmatic subjects was 2.62 with 95% confidence intervals ranging from 1.50 to 4.55. The associated p-value was small and suggested the probability of this odds ratio arising by chance was less than one in a thousand.

5.5.2 Strength

Generally speaking, the stronger an observed association the less likely it may be explained by confounding or potential bias in study design. In this study asthmatic patients attending a hospital out-patient clinic were two to three times more likely to live in a dwelling with evidence of damp than age and sex matched controls. Such odds ratios if true would be of clinical relevance, but are at a level where they could be significantly influenced by potential confounding factors such as socio-economic status.

As part of the statistical analysis of the study results, these odds ratios were adjusted for the potential influence of confounding factors such as unemployment, low household income and smoking status. The adjusted odds ratios differed little from the crude ratios implying little confounding effect. The possibility of unknown confounders however, can never be dismissed.

5.5.3 Consistency

If an observed association has been repeatedly found by different researchers in different locations, then it lends weight to the argument that it represents a true relationship. Our reported odds ratios for asthmatic subjects living in damp homes are similar to those in previous studies where associations were observed between prevalence of lower respiratory symptoms suggestive of asthma and damp in the home in countries such as Britain (131, 147), U.S.A. (137), Holland (139) Scandinavia (51, 144) and Canada (135-136). All these cross-sectional epidemiological studies found a higher prevalence of chronic respiratory symptoms in either adults or children living in damp or mouldy housing conditions, with reported odds ratios between 1.5 to 3.0. Often the highest odds ratios were for symptoms of cough and phlegm and lower odds ratios for asthma and wheeze (140).

The results of this study are also consistent with the few other case-control studies which have looked at the relationship between asthma and damp housing conditions. Such studies performed in Holland (100), Ireland (150), Nigeria (151), Kenya (68) and Sweden (152) all found a higher prevalence of damp or mould in the dwellings of asthmatic compared with control subjects, with odds ratios ranging between 2.0 to 11.0. The highest odds ratios reported were for damp or mould to be present in the bedrooms of asthmatic children.

The results of this case-control study are therefore consistent with those of other epidemiological studies reported in the literature, performed in both developing and industrialised countries. Such similar findings from different countries, studying different populations suggest a consistent association between asthma and living in damp or mouldy housing conditions.

5.5.4 Specificity

If the association between an environmental factor and a disease is specific, that is exposure to the risk factor under study will predispose to only one disease, then clearly there would be a strong argument in favour of causality. Such one-to-one relationships are however uncommon with the majority of diseases, including asthma, having a multi-factorial aetiology. It would be unreasonable to suggest that exposure to damp housing conditions was the sole environmental risk factor

for the development of asthma, but it is possible that such housing conditions either predispose to the development of asthma or exacerbate the severity of the disease. In short, the lack of specificity for the association under study does not exclude a causal relationship.

5.5.5 Temporality

Establishing a temporal relationship between damp housing and asthma from an observational study such as this is difficult. The question as to whether exposure to damp housing conditions predisposes to the development of asthma or whether asthmatic subjects are more likely to gravitate to poorer quality damp housing requires to be addressed.

Although this study has shown a consistent statistical correlation between asthma severity, patient disability and severity of damp in the home, all these variables were negatively associated with net household income. Patients with asthma were more likely to be unemployed or registered sick or disabled and have lower incomes. Such patients may gravitate towards poorer quality housing, or may not be able to afford to heat their home adequately. Such homes would be more prone to condensation and damp.

The majority of our patients lived in rented council accommodation and it is unlikely that patients with severe asthma were systematically allocated damp or mouldy housing. Central heating was installed in the majority of homes and its reported use in the two weeks prior to interview was unrelated to asthma severity. Multiple regression analysis confirmed these confounding variables to have little effect on the observed associations between asthma severity and severity of damp or mould in the home. It is possible that unemployed patients spending a greater proportion of their time indoors at home may have more prolonged exposure to the damp or mould in the home, which could adversely effect their asthma severity. The time spent indoors and hence exposure time to the adverse housing conditions was not estimated in this study.

Some of our patients developed their asthma whilst living in a previous dwelling to that surveyed. A sub-analysis of the data, restricted to only those subjects who developed asthma whilst living in their current dwelling, found a high proportion of asthmatics (68%) had damp detected by the surveyor. The asthmatic subjects

were more likely to have lived in a previous home with evidence of self-reported damp or mould than control subjects. These findings indirectly support the argument for a temporal relationship between damp housing and asthma. It is however possible that damp housing can adversely influence asthma severity, thus increasing the possibility of a patient requiring referral to a hospital out-patient clinic.

5.5.6 Biological gradient

If the association under investigation is one which can reveal a dose-response relationship, then any evidence for such a relationship would greatly strengthen the argument for causality. Previous epidemiological studies have not been able to convincingly demonstrate a dose-response relationship between damp housing conditions and either the prevalence of respiratory symptoms or severity of asthma. Platt *et al* suggested a dose-response relationship may exist between dampness severity in the home and the prevalence of non-specific symptoms in children and adults (146), but their findings were not conclusive.

None of the cross-sectional studies in which lung function measurements were taken were able to demonstrate a relationship between damp in the home and severity of airflow obstruction (137, 147-148). As subjects in these studies were recruited from the general population any of those with asthma were likely to have mild disease severity and therefore have relatively normal lung function. None of the previously reported case-control studies investigating the relationship between damp housing and asthma made any measurement of lung function or graded asthma severity. They were therefore unable to assess the presence of a dose-response relationship.

Chronic asthma severity can be judged by symptoms, medication requirements and objective measures of lung function. The latter are regarded as preferable as subjective assessment of symptoms by physicians and patients can be inaccurate (234). The trend to a more standardised, step-wise approach in pharmacologic therapy in asthma management implies the minimum treatment required to control symptoms can also serve as a marker of disease activity (235). Despite these considerations, there are currently no well validated measures of chronic asthma severity.

The asthma severity scoring system developed for use in this study was designed to incorporate all of the above features. It was felt that in the data analysis, an overall severity score would be more sensitive than independent use of each measure of asthma severity. Before being used, the proposed chronic asthma severity score was subjected to statistical tests of reliability as described in the methods section of this thesis.

The internal reliability of the severity score was supported by the high Cronbachs alpha and the results of the principal components analysis. These statistical tests suggested an excellent correlation between the individual components of the severity score and that each component made an acceptable contribution to the final score. The continuous severity scale therefore appeared to give a measure of physiological asthma severity. The severity score could also be collapsed to an ordinal scale to grade the asthmatic subjects into those with mild, moderate and severe disease.

Estimates as to the scores construct validity were obtained by comparing the final severity score with various indices of functional disease severity. Significant associations were observed between the severity score and the number of days spent off work or school with asthma, the patient's subjective assessment of their health and whether adults were regarded as unfit for employment because of their asthma.

By using this chronic asthma severity score and the measurements of exposure to damp or mould obtained during the survey, I attempted to demonstrate a dose-response relationship by two means; firstly to identify if the prevalence of damp and mould in the home increased with increasing severity of asthma and secondly, to identify if an increase in quantity of damp and mould in the home was associated with increasing asthma severity scores.

Trends were observed for an increase in the prevalence of both self-reported and surveyor confirmed damp in the dwellings of subjects with increasing severity of asthma. Control subjects and patients with mild asthma were likely to have a similar prevalence of damp in the home, whereas patients with moderate and severe asthma had almost twice the prevalence of damp as control subjects.

Significant statistical associations were observed between the quantity of damp and mould in the home and severity of asthma, including the asthma severity score and lung function measurements (FEV_1 percent and FEV_1/FVC ratio). The various methods employed to grade asthma severity all gave similar results. There

were statistically significant positive correlations between the total damp and mould scores for the dwelling and the asthma severity score ($r=0.30$). A significant negative correlation was observed between damp severity and FEV₁ percent ($r= -0.30$).

These results suggest a dose-response relationship between damp in the home and severity of asthma with the former accounting for approximately nine percent of the variance in the asthma severity score and degree of airflow obstruction.

5.5.7 Biological plausibility

It is often beneficial to the argument for causation if the suspected association is biologically plausible. There are several possible mechanisms to explain a higher prevalence of asthma in subjects living in damp dwellings. Increased exposure to house dust mites may result in increased sensitisation (83) or act as an exacerbating factor (85) for asthma and other allergic diseases. *Dermatophagoides pteronyssinus* thrives in damp conditions and both mite numbers and mite antigen concentrations are associated with higher indoor humidity (97, 205) and with indicators of damp in the home (97, 99).

The relationships between damp housing characteristics and house dust mite sensitivity in asthmatic subjects observed in this study are interesting. The prevalence of house dust mite sensitivity in asthmatic subjects was high (70%), but surprisingly, those subjects living in dry dwellings were more likely to be HDM sensitive than subjects living in homes with areas of damp. As HDM numbers are reported to be greater in dwellings with evidence of damp, the opposite finding would have been expected.

It is likely that house dust mite sensitivity in atopic subjects develops in early childhood. The lack of an association between house dust mite sensitivity in older children and adults and spot measurements of damp severity in the current home is therefore not surprising. It could also be argued that the more atopic subjects with asthma may have sought drier dwellings, giving rise to the above observation. The lack of information on HDM allergen levels in current and previous dwellings and the date of acquired house dust mite sensitivity of asthmatic subjects in this study does not allow further interpretation of this finding.

World-wide changes in housing conditions have meant in general homes are warmer and more humid, primarily as a result of better insulation. In some areas this has

been implicated in subjects developing allergy to the house dust mite where previously this was rare (176). Although damp dwellings are generally more humid and hence favour mite growth, focal areas of damp may create micro-environments optimising mite growing conditions. The sampling of house dust from mattresses or random areas of bedroom or living room floors undertaken in previous studies may under-estimate the potential risk of a subject's exposure to these allergens. House dust mite sampling was not performed in this study, but it could be argued that either targeting damp areas within the dwelling or airborne sampling may give a more representative measure of a subject's exposure to HDM allergens.

Although *D. pteronyssinus* is the commonest house dust mite in Britain, other mites are present in house dust, usually in smaller numbers (97). Some hydrophilic species including *Glycyphagus* spp, require higher relative humidities for optimum growth. Allergy to storage mites, which are antigenically different from the house dust mite and normally found in grain, hay and straw, is a recognised occupational risk among farmers (195).

Storage mites are found in significant numbers in house dust taken from damp dwellings (98, 195). Occupants of these dwellings are more likely to show allergic responses to these mites in the form of positive RAST and skin prick tests (197). The clinical relevance of this allergy in asthmatic subjects, with no relevant occupational history, is unclear.

Damp dwellings are also likely to have a higher prevalence of visible and hidden mould growth and therefore have higher levels of airborne fungal spores which can exacerbate respiratory symptoms in susceptible individuals (101). Between 10 - 15% asthmatic subjects have allergy to moulds, assessed by skin prick testing, most commonly to *Asp. fumigatus*, *Alt. Alternata*, *Penicillium* and *Cladosporium* (208-209, 236).

In one case-control study asthmatic subjects were more likely than matched controls to have mould within their dwelling and positive mould skin tests (208). However, a survey of 88 homes of children in Edinburgh found no correlation between visible mould growth and quantity of airborne spores. There was however a non-significant increase in total mould counts in the homes of children with significant exercise induced bronchoconstriction (149).

This study confirmed 17% asthmatic subjects had mould sensitivity on skin prick testing. All but one subject was classified as atopic on the basis of skin prick reactions

to other common allergens. Only those asthmatic subjects living in homes arbitrarily classified as having significant mould growth, had a greater prevalence of mould allergy. The number of subjects with mould allergy was however too small to allow meaningful comparisons between their asthma severity and housing characteristics.

It is possible that atopic subjects who are exposed to greater quantities of indoor mould are likely to develop mould sensitivity. Continued exposure to the mould might then have an adverse effect on their asthma, but evidence to support this hypothesis is lacking. Measurements of exposure to airborne moulds were not performed in this study. The relationships between the presence of damp, house dust mite allergen concentrations, visible mould growth and airborne mould spore counts could therefore not be examined.

The study results confirm that mould in a dwelling is likely to indicate the presence of severe damp, which in turn offers more favourable growing conditions for the house dust mite. The presence of visible mould growth in a dwelling may be associated with asthma only through this mechanism.

Subjects with mould allergy frequently have allergy to other allergens such as the house dust mite, as was observed in this study. The contribution of mould allergy to a subject's asthma is therefore difficult to assess, as bronchial provocation tests using mould spores are rarely performed. Further studies which quantify the exposure of asthmatic subjects to house dust mite allergens and to airborne moulds in damp homes are needed to address these issues.

5.5.8. Coherence

The cause and effect interpretation of the data should not seriously conflict with the known facts of the history and biology of the disease. It is widely accepted that asthma occurs in genetically predisposed individuals exposed to certain environmental trigger factors and that disease severity can be increased by constant exposure to high levels of aero-allergens.

The findings of this study are consistent with these views. Allergens, such as those associated with the house dust mite, occur in greater quantities in damp dwellings. It could be argued that susceptible individuals living in damp homes are more likely to develop asthma and that this may be more severe than if they had lived in a dry home.

5.5.9 Experiment

Occasionally it is possible to obtain experimental or semi-experimental evidence which can support the argument for a causality. If for example, because of an observed association some preventative action can be instituted, then the resultant effects on disease frequency and severity can be monitored. For instance, it has been shown that reduction of mite allergen in bedrooms, may modestly improve symptoms and medication requirements in mite-sensitive asthmatic subjects (237).

It follows, that if there were a causal relationship between damp housing and asthma, then action to reduce dampness in the home would be expected to favourably influence asthma morbidity. I am unaware of any study in the literature which has been designed to investigate this aspect of the proposed dose-response relationship. A small study set in North-west England found the installation of mechanical ventilation devices in homes were unable to lower indoor relative humidity to a level which would inhibit mite growth (238). The ventilation units did however improve condensation within the home and the effects of their installation in damp homes remains unclear.

5.5.10 The case for causality

The importance of adequate housing in improving and maintaining health has long been recognised. This study has confirmed living in damp or mouldy housing has an adverse effect on asthma severity, resulting in an increase in severity of airflow obstruction and patient disability. These findings appear to be fairly robust and the observed association between damp housing and asthma fulfils many of the criteria for a causal relationship as proposed by Bradford Hill. I have postulated that living in damp housing increases a subject's exposure to certain aero-allergens, in particular those relating to the house dust mite.

A reduction in exposure to the house dust mite can decrease bronchial hyperreactivity and improve asthma control (239-240). Asthma symptoms improve if patients live at high altitude, as there is an inverse relationship between altitude and numbers of house dust mites in dwellings (93). This is due to the unfavourable climatic conditions of lower temperatures and relative humidities at higher altitudes which prevent house dust mite growth.

A reduction in household humidity and correction of damp conditions, if practical, may therefore prove to be an effective measure of asthma control. If such a beneficial effect on asthma morbidity were to be demonstrated, the case for a causal relationship between damp housing and asthma would be further strengthened.

It is difficult to identify the means whereby an asthmatic subject could significantly reduce the quantity of damp and condensation within their dwelling. The cost of the necessary alterations to achieve this may be substantial. As many of the asthmatic subjects in this study were in low income households, they would be unlikely to afford these increased costs.

An alternative would be for the subject to seek a damp free home, perhaps with the same local housing authority. Although the results of this study suggest that such a move could improve a subject's asthma morbidity and reduce the degree of chronic airflow obstruction, perhaps by 10%, there is currently no study published in the literature to support this hypothesis. Until such time as this issue has been addressed, councils controlling local authority housing are unlikely to comply with such requests for re-housing.

In the long term, effective measures to reduce the risk of damp and condensation occurring in dwellings are required to be incorporated into future housing design. This could have the effect of reducing the considerable financial burden of asthma on the National Health Service and improve the quality of life of asthma sufferers and their families.

Chapter 6

Indications for further research

6.1 Confirmation of study findings

This study is the first to describe a dose-response relationship between severity of asthma and the severity of damp within the home. Further studies are required to confirm this observation. In this study, both the damp severity within the home and the degree of airflow obstruction were both measured at only one point in time. Serial markers of asthma severity such as daily peak expiratory flow rate monitoring and use of rescue bronchodilator therapy correlated with averaged measures of damp and mould within the subjects home over the same time period would allow a more robust examination of this association.

Subjects with more severe asthma often have higher degrees of bronchial hyperreactivity demonstrated by chemical challenge or exercise testing. The demonstration of a relationship between the degree of bronchial hyper-responsiveness and severity of damp in the home would give further objective evidence to support the argument for the proposed dose-response relationship observed in this study.

6.2 Allergen exposure in damp homes

It is postulated that subjects living in damp dwellings have more severe asthma because they are exposed to higher concentrations of aero-allergens, particularly those arising from the house dust mite and various indoor moulds. Further research is required to explore and confirm this hypothesis.

Whilst many studies have correlated asthma severity with quantities of *Der p I* in house dust, the relationship between asthma severity and airborne *Der p I* is less certain. Airborne *Der p I* levels have not yet been correlated with severity of damp in dwellings. The relationships between dampness severity, *Der p I* levels in house dust and airborne *Der p I* require further investigation.

The presence of visible mould growth in dwellings has been associated with asthma, but there is a poor correlation between visible mould growth and airborne mould spore concentrations. No study has yet demonstrated a correlation between indoor mould spore concentrations and severity of asthma. This may reflect the difficulty in quantifying indoor mould spore exposure, whether the association is in part due to respondent bias or whether visible mould growth is merely acting as a confounder. Mould growth in a dwelling may only indicate the presence of more severe damp

and hence more favourable growing conditions for the house dust mite. The inter-relationships between dampness severity, house dust mite numbers, visible mould growth and airborne mould spore concentrations requires elucidation.

6.3 Improving dampness within the home

The results of this study suggest that improving living conditions and in particular reducing the quantity of damp in the home may improve asthma morbidity. There are four methods by which exposure to damp may be reduced.

1. Reduction in moisture production

We found little difference in moisture production activities in the dwellings of asthmatic and control subjects. General advice such as using an electric cooker and drying clothes either outside or using a vented tumble drier may help reduce condensation in the dwelling. There is no evidence in the literature to support giving such advice to patients with asthma.

2. Installation of portable dehumidifiers

These devices have previously been regarded as too small and lacking the power to significantly reduce indoor relative humidity (193). With improving technology, these units have become smaller and more efficient and their manufacturers are making bolder statements about their beneficial effects. Although the manufacturers usually recommend the dehumidifier is positioned centrally within the house, it may be more effective at reducing humidity and house dust mite numbers if placed within the bedroom. There is no evidence to support the claims that they have any beneficial effect on asthma morbidity, or even influence house dust mite numbers and this requires investigation.

3. Improving the fabric of the building

Dampness in dwellings due to poor building fabric such as leaking roofs, rising damp from a breached damp course or penetrating damp due to poor brickwork are more common in older dwellings and are difficult and costly to

repair. These causes of damp are relatively rare and the majority of damp in dwellings occurs as a result of condensation. Efforts to reduce condensation could therefore reduce the prevalence of damp and mould.

Besides reducing moisture production in the dwelling as discussed above, condensation is prevented by increasing both the indoor temperature and degree of ventilation. These are not easily achieved goals in typical British housing. Besides, in the present state of technology, the initial installation costs and the running costs of these condensation remedies may be prohibitively high.

4. Re-housing in dry accommodation

If living in a damp dwelling contributes to an increase in asthma severity as described in this study, then the re-housing of asthmatic subjects in dry accommodation would be expected to reduce asthma morbidity. If confirmed this would provide further evidence to support a dose-response relationship between damp housing and asthma. Such a study may remain beyond the scope of a clinical trial.

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Appendices A

A Questionnaire

Record number	01	1-2
Sample	<input type="text"/>	3
Respondent number	<input type="text"/> <input type="text"/> <input type="text"/>	4-6
Interviewer	<input type="text"/> <input type="text"/>	7-8
Date of interview	<input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/>	9-12

Govan Health Survey

Household structure

- 1 a) Could you tell me how many people are living here permanently at the moment?
 b) What relation are they to you?
 c) What age are they?

Record information on grid below. The first line of information recorded should be for the subject (the adult respondent or the child)

	Relation to informant	Age	Sex
1			
2			
3			
4			
5			
6			
7			
8			

INTERVIEWER RECORD

number of adults (aged 16 + years)	<input type="checkbox"/>	13
number of children aged 0-4 years	<input type="checkbox"/>	14
number of children aged 5-11 years	<input type="checkbox"/>	15
number of children aged 12-15 years	<input type="checkbox"/>	16
total number in household	<input type="checkbox"/> <input type="checkbox"/>	17-18

a) Whether respondent is the subject or the parent of a child subject

adult subject	<input type="checkbox"/>	1
parent of child subject	<input type="checkbox"/>	2
		19

b) Whether respondent is male or female.

male	<input type="checkbox"/>	1
female	<input type="checkbox"/>	2
		20

c) If a child is the subject: whether male or female

male	<input type="checkbox"/>	1
female	<input type="checkbox"/>	2
		21

d) Age of subject

<input type="checkbox"/> <input type="checkbox"/>	22-23
---	-------

Health status: asthma and illness history

These questions relate to the health of the target subject. If the subject is an adult then they refer to the health of the respondent. When the target subject is a child (aged 15 years or less), the questions refer to the child's health.

Can I ask you a few questions about your [child's name's] health. Some of the questions will be rather general but others will be about specific symptoms or problems.

- 2 Do you [does *child's name*] or have you [has *child's name*] ever woken up first thing in the morning with: *[tick all that apply]*

wheeze	<input type="checkbox"/>	1	24
a feeling of tightness in your chest	<input type="checkbox"/>	1	25
cough	<input type="checkbox"/>	1	26

- 3 Are you [is...] or have you [has...] ever been woken up during the night by:

wheeze	<input type="checkbox"/>	1	27
a feeling of tightness in your chest	<input type="checkbox"/>	1	28
cough	<input type="checkbox"/>	1	29

If NONE of these symptoms morning or night: GO TO Q5

- 4 How often do or did you [...] usually have these problems? *[Record most frequent]*

less than once a week	<input type="checkbox"/>	1	
2 - 3 times a week	<input type="checkbox"/>	2	
4 - 5 times a week	<input type="checkbox"/>	3	
all or most days/nights	<input type="checkbox"/>	4	30

- 5 Do you [does.....] have attacks of breathlessness during the day when doing any of the following activities? Does this happen every or most times, sometimes, or never?

Go through list, asking for each activity whether respondent (or child) experiences breathlessness

	Every time	Some- times	Never	
playing sports/ games	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	31
walking up hills	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	32
walking up a flight of stairs	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	33
walking outside on the level	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	34
walking around the house	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	35
getting washed or dressed	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	36
sitting or lying still	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	37

6 Do you [does] ever use a nebuliser?

yes ☐ 1
no ☐ 2

38

7 Do you [does] ever use inhaled medicines (inhalers, puffers)?

yes ☐ 1
no ☐ 2 → Go to Q12

39

8 Which of these inhalers do you [does.....] use? (Show card 1 and tick all that apply)

Ventolin	<input type="checkbox"/>	1	40
Aerolin autoinhaler	<input type="checkbox"/>	1	41
Bricanyl	<input type="checkbox"/>	1	42
Duovent	<input type="checkbox"/>	1	43

If yes to any:

9 a) Do you [does.....] use the inhaler whenever you need to or as prescribed?

when needed	<input type="checkbox"/>	1	
as prescribed	<input type="checkbox"/>	2	
other.....	<input type="checkbox"/>	3	44

b) And, how often do you [does....] usually use this/these inhaler(s) then?

less than once a week	<input type="checkbox"/>	1	
1 - 3 times a week	<input type="checkbox"/>	2	
4 - 6 times a week	<input type="checkbox"/>	3	
7 or more times a week/daily	<input type="checkbox"/>	4	45

10 Do you [does.....] use any of the following inhalers? (Show card 2 and, if necessary, check on bottle for dosage in micrograms)

Becotide	<input type="checkbox"/>	1	46
Becloforte	<input type="checkbox"/>	1	47
Pulmicorte	<input type="checkbox"/>	1	48
Intal	<input type="checkbox"/>	1	49
other.....	<input type="checkbox"/>	1	50

11 How many puffs a day would you [.....] usually have?

CALCULATE TOTAL DAILY DOSE: Puffs X dose
[Check dose with patient or on medication bottle]

51-54

- 12 In the past 12 months, how many courses (if any) of Prednisolone (steroid) tablets have you taken for a chest complaint? *[exclude Prednisolone course taken for other conditions]*

none	<input type="checkbox"/>	0	
1 - 2 courses	<input type="checkbox"/>	1	
3 or more courses (but not all the time)	<input type="checkbox"/>	2	
constant/continuous/all the time	<input type="checkbox"/>	3	55

- 13 Are you [.....] on any other regular prescribed medication?

not on any other regular medication	<input type="checkbox"/>	0	56
on regular medication	<input type="checkbox"/>	1	

- 14 *If yes:* What do you [.....] take? What is/are they for?

Write down name of prescribed medications

1.....	<input type="checkbox"/>	<input type="checkbox"/>	57-58
2.....	<input type="checkbox"/>	<input type="checkbox"/>	59-60
3.....	<input type="checkbox"/>	<input type="checkbox"/>	61-62
4.....	<input type="checkbox"/>	<input type="checkbox"/>	63-64

CHECK BACK TO Q2, Q3 and Q5: If respondent has answered yes to any of the items about cough, wheeze, or feelings of tightness in their chest (first thing in the morning and/or during the night) answer the following questions. All others GO TO Q26 (MRC Bronchitis Scale)

- 15 How old were you [....] when you first noticed these symptoms? ☐☐ 65-66

- 16 So, how long has it been since these symptoms first began? *(Only code weeks or months if symptoms are reported as beginning recently: otherwise leave blank)*

years	<input type="checkbox"/>	<input type="checkbox"/>	67-68
months	<input type="checkbox"/>	<input type="checkbox"/>	69-70
weeks	<input type="checkbox"/>	<input type="checkbox"/>	71-72

- 17 In the past 12 months, have you [.....] had any time off work [school] because of these problems? *If not working/not at school: Have you [....] had times when you [.....] have had to stay indoor or have been confined to the house or bed because of these problems*

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	73

- 18 *If yes:* How many days have you [....] had to take off/stay in bed/ stay inside? ☐☐ 74-75

Record number	02	1-2
Sample	<input type="checkbox"/>	3
Respondent number	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	4-6

COMMUNITY SUBJECTS ONLY (all others GO TO Q26)

19 Have you (or your parents) ever consulted a GP about these problems?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	→ Go to Q22
uncertain	<input type="checkbox"/>	3	7

20 Have you consulted your doctor (has your doctor been consulted) about these problems in the last 12 months?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	8

21 If yes: How many times in the past 12 months?

<input type="checkbox"/> <input type="checkbox"/>	9-10
---	------

22 Have you [.....] ever been referred to a specialist at a hospital for these problems?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	→ Go to Q25

23 If yes: In the past 12 months, have you [.....] received treatment from a specialist at a hospital as an in-patient or as an out-patient for these problems?

no, neither	<input type="checkbox"/>	0	
out-patient only	<input type="checkbox"/>	1	
in-patient only	<input type="checkbox"/>	2	
both	<input type="checkbox"/>	3	12

24 If in-patient episode: How many times in the past year have you [....] been admitted for these problems?

<input type="checkbox"/> <input type="checkbox"/>	13-14
---	-------

25 Has any doctor ever told you that they think you [.....] have asthma?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	
uncertain	<input type="checkbox"/>	3	15

ALL RESPONDENTS
MRC Bronchitis Scale

26 Do you [....] usually cough first thing in the morning?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	16

27 Do you [.....] usually cough during the day or night in winter?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	17

If yes to Q26 or Q27:

28 Do you [.....] cough like this on most days for as much as 3 months each year?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	18

29 Do you [.....] usually bring up phlegm (spit from the chest) first thing in the morning?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	19

30 Do you [....] usually bring up phlegm during the day or at night in winter?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	20

If yes to Q29 or Q30:

31 Do you bring up phlegm most days for as much as 3 months each year?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	21

32 In the past 12 months have you had a period of cough or phlegm lasting 3 weeks or more?

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	22

General health

33 Overall, how would you describe your [.....] health in the past 2 weeks? Would you say it was:

excellent	<input type="checkbox"/>	1	
good	<input type="checkbox"/>	2	
fair	<input type="checkbox"/>	3	
poor	<input type="checkbox"/>	4	
very poor	<input type="checkbox"/>	5	23

Housing: conditions, facilities, and history

34 How long have you lived at this address?

Years Months

24-27

35 Did you have to move from your last house or was it your own choice?

forced move ☐ 1
 voluntary move ☐ 2
 bit of both ☐ 3

28

36 What were the reasons for your move? *[If necessary: PROMPT]* Were there ever any problems with: Tick the relevant boxes: if none of these mentioned leave blank.

problems with the area (too rough/noisy, neighbours) ☐ 1 29
 dampness and/or condensation ☐ 1 30
 other problems connected with the house (too large/ small/ high) ☐ 1 31
 personal reasons, not connected with health ☐ 1 32
 personal reasons, connected with health ☐ 1 33

ONLY IF PREVIOUS PROBLEMS WITH DAMPNESS IN HOUSING ARE MENTIONED

37 How long did you live in your last house/flat?

Years Months

34-37

38 How serious was the dampness problem in your last house/flat: would you say it had been a serious problem or more of a nuisance than a problem?

A serious problem ☐ 1
 More of a nuisance ☐ 2
 Can't say/ don't know/remember ☐ 3

38

39 Was there ever mould growth on the walls?

yes ☐ 1
 no ☐ 2
 uncertain/don't know/remember ☐ 3

39

ALL RESPONDENTS

- 40 Have you ever lived in any (other) houses or flats where dampness or mould growth was a problem? *(Do not include current/present house)*

yes	<input type="checkbox"/>	1	
no	<input type="checkbox"/>	2	
uncertain/don't know/remember	<input type="checkbox"/>	3	40

- 41 Was your last house/flat privately owned or was it rented?
If rented: Who was it rented from?

	Last house	
privately owned	<input type="checkbox"/>	1
rented from the Council	<input type="checkbox"/>	2
rented from Scottish Homes	<input type="checkbox"/>	3
rented from housing association/co-operative	<input type="checkbox"/>	4
privately rented	<input type="checkbox"/>	5
other.....	<input type="checkbox"/>	6
		41

- 42 What about this house: is it privately owned or rented?
If rented: Who is it rented from?

	Current house	
privately owned	<input type="checkbox"/>	1
rented from the Council	<input type="checkbox"/>	2
rented from Scottish Homes	<input type="checkbox"/>	3
rented from housing association/co-operative	<input type="checkbox"/>	4
privately rented	<input type="checkbox"/>	5
other.....	<input type="checkbox"/>	6
		42

- 43 I'd like to ask you some questions about this house/flat?

- a) What rooms do you have? How many of ... do you have? *[Specify below]*
b) Are there any rooms which you have to use but would prefer not to use? *[Specify below]*
c) Are there any rooms you cannot use? *[Specify below]*

<u>Type of room</u>	Number	Have to use	Can't use	
bedsitting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	43-45
bedroom	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	46-48
living	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	49-51
kitchen	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	52-54
bathroom	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	55-57

If any rooms "have to" or can't use:

44 a) Why is that? [Prompt for each reason below and tick all that apply]

Reasons for reluctant/non-use

coldness	<input type="checkbox"/>	1	58
damp	<input type="checkbox"/>	1	59
damage	<input type="checkbox"/>	1	60
safety	<input type="checkbox"/>	1	61
noise	<input type="checkbox"/>	1	62
other.....	<input type="checkbox"/>	1	63

b) Does this cause any of these problems? [Show card 3 and check for each problem. Tick all that apply]

Problems caused

overcrowding	<input type="checkbox"/>	1	64
arguments/bad feeling	<input type="checkbox"/>	1	65
financial problems	<input type="checkbox"/>	1	66
problems having people round	<input type="checkbox"/>	1	67
emotional upset	<input type="checkbox"/>	1	68
other.....	<input type="checkbox"/>	1	69

45 What kind of cooker (oven) do you mostly use?

Gas	<input type="checkbox"/>	1	
Electric	<input type="checkbox"/>	2	
Dual fuel	<input type="checkbox"/>	3	
Paraffin	<input type="checkbox"/>	4	
Microwave	<input type="checkbox"/>	5	
Other.....	<input type="checkbox"/>	6	
None	<input type="checkbox"/>	0	70

Record number 03 1-2
 Sample ☐ 3
 Respondent number ☐☐☐ 4-6

46 How is this flat/house heated?

- a) What heating is available?
 b) What forms of heating have been used in the past 2 weeks?

Show card 4 and check for each type of heating

	Available	Used in past 2 weeks	
central heating radiators (gas or elec)	<input type="checkbox"/> 1	<input type="checkbox"/> 1	7-8
storage heaters	<input type="checkbox"/> 1	<input type="checkbox"/> 1	9-10
gas fires	<input type="checkbox"/> 1	<input type="checkbox"/> 1	11-12
electric fires	<input type="checkbox"/> 1	<input type="checkbox"/> 1	13-14
coal/solid fuel	<input type="checkbox"/> 1	<input type="checkbox"/> 1	15-16
portable gas or paraffin heaters	<input type="checkbox"/> 1	<input type="checkbox"/> 1	17-18
Other.....	<input type="checkbox"/> 1	<input type="checkbox"/> 1	19-20

47 How has the family washing mostly been done during the past 2 weeks?

Washing machine at home	<input type="checkbox"/> 1	
By hand	<input type="checkbox"/> 2	
Launderette	<input type="checkbox"/> 3	
Elsewhere	<input type="checkbox"/> 4	21

48 Where has the family washing been done during the past 2 weeks?

Kitchen	<input type="checkbox"/> 1	
Bathroom	<input type="checkbox"/> 2	
Utility room	<input type="checkbox"/> 3	
Elsewhere	<input type="checkbox"/> 4	22

49 On average, how often has the family washing been done in the last 2 weeks?

Times in
 past 2 weeks
☐☐ 23-24

50 What method has mostly been used to dry washing in the past 2 weeks?

Drying machine: vented	<input type="checkbox"/> 1	
Drying machine: unvented	<input type="checkbox"/> 2	
Left to dry inside (over radiator/clothes horse etc)	<input type="checkbox"/> 3	
Hung outside	<input type="checkbox"/> 4	25

51 Where have the clothes/washing been dried? [tick all that apply]

Kitchen	<input type="checkbox"/>	1	26
Bathroom	<input type="checkbox"/>	1	27
Bedroom	<input type="checkbox"/>	1	28
Utility room	<input type="checkbox"/>	1	29
Outside	<input type="checkbox"/>	1	30
Elsewhere	<input type="checkbox"/>	1	31

52 a) Is condensation a problem in your home? Not just normal condensation on windows? Is it a serious problem or more of a nuisance than a problem?

b) What about dampness: is that a problem in your home? Is it a serious problem or more of a nuisance than a problem?

	Condensation	Dampness	
A serious problem	<input type="checkbox"/> 1	<input type="checkbox"/> 1	
More of a nuisance than a problem	<input type="checkbox"/> 2	<input type="checkbox"/> 2	
Not a current problem	<input type="checkbox"/> 0	<input type="checkbox"/> 0	
In the past only (in this house)	<input type="checkbox"/> 3	<input type="checkbox"/> 3	32 33

53 If no: Have these ever been a problem in this house? [If yes: code 3 above]

54 Are there times in the winter when you can't keep your house warm enough?
If yes: Is that most of the time, quite often or only occasionally?

most of the time	<input type="checkbox"/>	1	
quite often	<input type="checkbox"/>	2	
only occasionally	<input type="checkbox"/>	3	
never	<input type="checkbox"/>	4	34

55 Do you keep any pets in the house? If yes: What pets do you have? [Tick all that are mentioned]

Cat(s)	<input type="checkbox"/>	1	41
Dog(s)	<input type="checkbox"/>	1	42
Hamster/gerbil/mice/rabbit/guinea pig	<input type="checkbox"/>	1	43
Budgie or other bird(s)	<input type="checkbox"/>	1	44
Other pet.....	<input type="checkbox"/>	1	45

Cigarette smoking in the household

56 Does any adult in the house smoke cigarettes at the present time?

yes

☐ 1

no

☐ 2

→ Go to Q58

46

57 a) Who in the household is a current smoker? *[tick all that apply]*

self/respondent

☐ 1

47

husband/wife/partner

☐ 1

48

other adult(s)

☐ 1

49

b) So how many adults altogether are current smokers?

number of current adult smokers in household (none = 0)

☐

50

58 Has anyone in the household given up smoking?

yes

☐ 1

no

☐ 2

51

59 If yes: a) Who? and b) When was that?

	Self	Partner	Other adult	
gave up in past 6 months	<input type="checkbox"/> 1	<input type="checkbox"/> 1	<input type="checkbox"/> 1	
gave up in past year	<input type="checkbox"/> 2	<input type="checkbox"/> 2	<input type="checkbox"/> 2	
gave up more than a year ago	<input type="checkbox"/> 3	<input type="checkbox"/> 3	<input type="checkbox"/> 3	52 53 54

IF RESPONDENT IS A CURRENT CIGARETTE SMOKER

60 How many cigarettes did you smoke yesterday?

1 - 5

☐ 1

6 - 10

☐ 2

11 - 15

☐ 3

16 - 20

☐ 4

21 - 30

☐ 5

31 - 40

☐ 6

40 +

☐ 7

55

61 If parent of child subject: To the best of your knowledge, does [name of child] smoke cigarettes?

yes

☐ 1

no

☐ 2

uncertain

☐ 3

56

Social characteristics

62 Can you tell me if you are married or living with a partner, single, divorced, separated or widowed?

married/living with a partner	<input type="checkbox"/>	1	
single	<input type="checkbox"/>	2	
separated	<input type="checkbox"/>	3	
divorced	<input type="checkbox"/>	4	
widowed	<input type="checkbox"/>	5	57

I would like to find out how all the adults (aged 16 +) in the house are occupied during the day.

63 Are all the adults currently in paid employment?

yes	<input type="checkbox"/>	1 → Go to Q67	
no	<input type="checkbox"/>	2	58

If no:

64 a) Who is not in paid employment at the moment?

respondent/parent of child	<input type="checkbox"/>	1	59
partner	<input type="checkbox"/>	1	60
adult 1	<input type="checkbox"/>	1	61
adult 2	<input type="checkbox"/>	1	62
adult 3	<input type="checkbox"/>	1	63
adult 4	<input type="checkbox"/>	1	64

b) Are you/your partner/other adults currently looking for work?

	Respondent	Partner	Other adult(s)	
yes	<input type="checkbox"/> 1	<input type="checkbox"/> 1	<input type="checkbox"/> 1	
no	<input type="checkbox"/> 2	<input type="checkbox"/> 2	<input type="checkbox"/> 2	65 66 67

If no for self or partner:

65 Which of the following best describes your (and your husband/wife/partner's) position at the moment: [Show card 5]

	Self	Partner	
Looking for work	<input type="checkbox"/> 1	<input type="checkbox"/> 1	
Temporarily sick/ill	<input type="checkbox"/> 2	<input type="checkbox"/> 2	
Permanently sick/disabled	<input type="checkbox"/> 3	<input type="checkbox"/> 3	
Full-time student	<input type="checkbox"/> 4	<input type="checkbox"/> 4	
Housewife	<input type="checkbox"/> 5	<input type="checkbox"/> 5	
Retired	<input type="checkbox"/> 6	<input type="checkbox"/> 6	
Other.....	<input type="checkbox"/> 7	<input type="checkbox"/> 7	68 69

If respondent or partner is unemployed and looking for work:

66 How long have you [your partner] been unemployed?

	Respondent	Partner
less than one month	<input type="checkbox"/> 1	<input type="checkbox"/> 1
1 - 3 months	<input type="checkbox"/> 2	<input type="checkbox"/> 2
4 -6 months	<input type="checkbox"/> 3	<input type="checkbox"/> 3
7 - 12 months	<input type="checkbox"/> 4	<input type="checkbox"/> 4
more than a year	<input type="checkbox"/> 5	<input type="checkbox"/> 5

72 73

67 What is your current (most recent) occupation [job]?

68 What about your husband/wife/partner: What is his/her current (most recent) occupation [job]?

69 Could you look at this card *[show card 6]*, and tell me how much money there is coming into the house each week, after deductions for tax and insurance? Which number best represents your income? *[INCLUDE AMOUNTS PAID DIRECTLY BY DSS FOR HEATING AND RENT]*

01 < £20	08 £141 - £160
02 £21 - £40	09 £161 - £180
03 £41 - £60	10 £181 - £200
04 £61 - £80	11 £201 - £250
05 £81 - £100	12 £251 - £300
06 £101 - £120	13 £301 +
07 £121 - £140	

☐ ☐

74-75

70 About how much of the weekly household income is spent on:

a) Food

☐ ☐

76-77

b) Heating

☐ ☐

78-79

Appendices B

A publication arising from the study.

Damp housing and asthma: a case-control study

I J Williamson, C J Martin, G McGill, R D H Monie, A G Fennerty

Abstract

Background – Several epidemiological studies have reported a higher prevalence of respiratory symptoms in subjects living in damp housing, but links with specific respiratory diseases such as asthma have not been satisfactorily established.

Methods – One hundred and two subjects with physician diagnosed asthma and 196 age and sex matched controls were interviewed; 222 (75%) then agreed to have their dwelling surveyed for dampness. The prevalence of both self-reported and observed dampness in the homes of the asthmatic subjects and controls were compared. Both asthma and the severity of the dampness were quantified so that the possibility of a dose-response relationship could be investigated.

Results – Asthmatic subjects reported dampness in their current (odds ratio (OR) 1.92, 95% confidence interval (CI) 1.18 to 3.12) and previous (OR 2.11, 95% CI 1.29 to 3.47) dwellings more frequently than control subjects. The surveyor confirmed dampness in 58 of 90 (64%) dwellings of asthmatic subjects compared with 54 of 132 (41%) dwellings of control subjects (OR 2.62, 95% CI 1.50 to 4.55). This association persisted after controlling for socioeconomic and other confounding variables (adjusted OR 3.03, 95% CI 1.65 to 5.57). The severity of asthma was found to correlate statistically with measures of total dampness ($r=0.30$, $p=0.006$) and mould growth ($r=0.23$, $p=0.035$) in the dwelling. Patients living in homes with confirmed areas of dampness had greater evidence of airflow obstruction than those living in dry homes (mean difference in forced expiratory volume in one second (FEV₁) 10.6%, 95% CI 1.0 to 20.3).

Conclusions – Asthma is associated with living in damp housing and there appears to be a dose-response relationship. Action to improve damp housing conditions may therefore favourably influence asthma morbidity.

(Thorax 1997;52:229-234)

Keywords: asthma, damp housing.

possibility that damp housing may adversely affect health and, in particular, predispose to respiratory symptoms has been the focus of several recent cross-sectional epidemiological studies.⁵⁻¹³ All reported a higher prevalence of respiratory symptoms, especially wheeze, in subjects living in damp housing, but links with specific respiratory diseases such as asthma could not be satisfactorily established. A few studies of case-control design do, however, suggest that asthmatic subjects are more likely to live in homes with evidence of dampness with the highest odds ratios for children sleeping in damp bedrooms.¹⁴⁻¹⁶

Several biologically plausible mechanisms could account for such an association. Asthmatic subjects are frequently allergic to house dust mite (HDM) and moulds, both of which are found in greater numbers in damp dwellings.¹⁷⁻²⁰ As higher levels of exposure to HDM allergen are known to increase asthma severity,¹² it is theoretically possible for damp housing to influence the severity of asthma adversely. No such relationship has yet been demonstrated.

Establishing a link between asthma and damp housing is by no means straightforward. The design of such a study has to eliminate many areas of potential bias. If information on health and measures of dampness in the dwelling is obtained from the same questionnaire, reporting or recall bias may occur with the potential for subjects in damp dwellings to over-report or exaggerate the severity of the symptoms.²¹⁻²³ This has been a particular criticism of many of the previous studies and it is universally agreed that objective measurements of dampness are therefore preferable. Respiratory health and dampness of housing may also be indirectly related through socioeconomic status, and particular attention has to be given to confounding factors.

This case-control study was designed, firstly, to establish whether subjects with physician diagnosed asthma were more likely than age and sex matched controls to live in damp housing and, secondly, to determine whether living in such conditions adversely influences the severity of asthma.

Methods

SUBJECTS

Consecutive patients with physician diagnosed asthma aged 5-44 years attending the Southern General Hospital Asthma Clinic between November 1992 and February 1993 were eligible for entry into the study. Three patients refused to participate and 102 were recruited. For each asthmatic patient entered, two control subjects matched for sex and age to within five years were randomly selected from the Greater Glas-

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Received 2 February 1996
Returned to authors
23 May 1996
Revised version received
21 October 1996
Accepted for publication
7 November 1996

Damp housing is a common problem in Britain today. National surveys have estimated that between 25% and 33% of dwellings are affected to some degree by either dampness, condensation, or mould growth.¹⁻³ The problem tends to be greatest in inner city housing where evidence of disrepair due to dampness has been found in as many as 47% of dwellings.¹ The

gow Health Board Community Health Index. If a selected control subject was no longer resident at the contact address or refused to participate, a further matched control was selected as a replacement. Two hundred and one of the 450 subjects randomly selected from the index were no longer resident at the contact address and could not be traced; 196 (79%) control subjects successfully contacted agreed to participate in the study. All asthmatic and control subjects lived within the catchment area of the hospital defined by area postal codes G51-53.

Approval for the study was obtained from the local ethics committee.

QUESTIONNAIRE

All subjects completed a structured interview with a trained researcher. The questionnaire was a modified version of that used by Martin *et al* in two previous studies investigating the relationship between respiratory symptoms and damp housing.^{5,22} Questions relating to housing conditions included the presence of current dampness or condensation in the home and exposure to dampness and mould in previous dwellings. Questions regarding respiratory symptoms included the presence, frequency and severity of wheeze, chest tightness, cough, and shortness of breath on exercise. Current asthma medications and the number of exacerbations of asthma requiring oral steroids in the previous year were noted.

LUNG FUNCTION

Patients performed spirometric tests (Vitalograph) at the asthma clinic at the time of entry to the study. The best forced expiratory volume in one second (FEV₁) and the ratio of FEV₁ to the forced vital capacity (FVC) were recorded from three attempts. FEV₁ was expressed as a percentage of the predicted values.

ASTHMA SEVERITY SCORE

An asthma severity score was calculated for each patient based on questionnaire responses regarding severity of asthma symptoms, medication requirements, and FEV₁ recorded at the last clinic visit (table 1). The sum of these seven severity items comprised the asthma severity raw score, with a possible range of 0 to 24. Asthmatic subjects were divided into approximately three equal sized groups labelled mild (raw severity score 0-8), moderate (9-13), and severe (14-24).

DAMPNESS SURVEY

After completion of the questionnaire, subjects were asked if their home could be independently assessed for dampness and mould by a qualified surveyor who would be unaware of their health status. Each dwelling was surveyed in a standard fashion as detailed below.

1. Spot temperature and relative humidity measurements were recorded outdoors and

Table 1 Asthma severity score

Severity variable	Score	n
Frequency of asthma symptoms		
Never	0	0
Less than once per week	1	29
2-3 times per week	2	28
4-5 times per week	3	9
Most days or nights	4	36
Shortness of breath on exercise		
Never	0	8
Playing games or sports	1	4
Walking uphill or stairs	2	39
Walking on level	3	15
Walking around house or dressing	4	36
Inhaled β_2 agonist use		
Never	0	2
Less than daily	1	35
Daily	2	65
Dose of inhaled steroid		
None	0	9
Up to 400 μ g daily (or Intal)	1	25
401 to 800 μ g daily	2	15
801 to 1500 μ g daily	3	35
>1500 μ g daily	4	18
Nebuliser use		
No	0	84
Yes	2	18
Use of oral steroids in last 12 months		
None	0	44
1 or 2 short courses	1	29
More than 2 courses	2	25
Maintenance	4	4
Lung function: FEV ₁ (%predicted)		
>80%	0	57
70-79%	1	11
60-69%	2	14
50-59%	3	6
<50%	4	9
Not measured	—	5

within each room in the dwelling using a Whatman R 200 digital hygrometer.

2. An electronic resistance type moisture meter (Protimeter Surveymaster) was used to obtain measurements of dampness just above skirting board height from three points on each wall (usually the middle and either end) in every room in the dwelling. At each point where a measurement was obtained dampness was graded semi-quantitatively, depending on the percentage scale deflection on the meter, as 0 (dry, <10%), 1 (11-25%), 2 (26-50%), 3 (51-75%), and 4 (>76%). The sum of all these dampness measurements (total dampness score) and the worst grade of dampness recorded were used as measures of the severity of dampness for each dwelling.

3. The presence and severity of visible mould growth on each wall in each room of the dwelling was graded subjectively on a four-point scale where 0=absent, 1=trace, 2=obvious but localised, and 3=obvious and widespread. The sum of these grades (total mould score) was used as a measure of mould severity within the dwelling. Dwellings with a total mould score of 3 or more were classified as having significant mould.

STATISTICAL ANALYSIS

Asthmatic and control subjects were compared in groups rather than individual matched pairs. Comparisons between categorical groups were made using the χ^2 test with odds ratios and their 95% confidence intervals or χ^2 trend stated where appropriate. Continuous variables were compared using the Student's *t* test or Wilcoxon sum rank test. The dampness and mould severity scores were positively skewed and logarithmic transformations were used for

Table 2 Comparison of sociodemographic characteristics between asthmatic and control subjects

	Asthmatic (n = 102)	Control (n = 196)	p value
Mean (SD) age (years)	26.4 (12)	25.8 (12)	NS
Male sex	36 (35)	78 (40)	NS
Mean (SD) persons/room	0.65 (0.23)	0.65 (0.23)	NS
Employment status*			
Employed	36 (35)	108 (55)	0.001
Unemployed	5 (5)	23 (12)	
Sickness benefit	35 (34)	16 (8)	
Housewife	18 (18)	41 (21)	
Other	8 (8)	8 (4)	
No adult in paid employment	47 (46)	70 (36)	NS
Housing tenure			
Rented	67 (66)	108 (55)	NS
Mean (SD) duration of tenancy (years)	7.4 (6.5)	7.4 (7.0)	NS
Net weekly household income			
Less than £200	76 (75)	118 (60)	0.02
Heating			
Central	67 (66)	121 (62)	NS
Gas fire	63 (62)	90 (46)	0.01
Paraffin	8 (8)	12 (6)	NS
Gas cooking	66 (65)	112 (57)	NS
Clothes dried indoors	71 (70)	137 (70)	NS
Respondent smoking status			
Non-smoker	48 (47)	82 (42)	0.001
Ex-smoker	25 (24)	18 (9)	
Smoker	29 (29)	96 (49)	
Smoker in household	60 (59)	137 (70)	0.05
Household pet	57 (56)	90 (46)	NS

Figures represent number (percentage) unless otherwise stated. p values are calculated from χ^2 test or Student's *t* test.

* Employment status of adult respondent or parent of child.

statistical analysis. Linear associations between continuous variables were assessed using the Pearson correlation coefficient. When controlling for confounding, if the outcome variable was binary, multiple unconditional logistic regression models were constructed using the maximum likelihood method with adjusted odds ratios and their 95% confidence intervals calculated from the logistic regression coefficients, otherwise multiple linear regression models were constructed using the stepwise method. Analysis was performed using the SPSS for Windows, Release 6.0.²⁴ A significance level of 5% was used.

Results

DEMOGRAPHIC CHARACTERISTICS

Two hundred and ninety eight subjects (102 with asthma and 196 controls) were successfully recruited. A summary of their demographic characteristics is listed in table 2. Although control subjects were matched for age and sex, several differences were identified between the two groups, particularly relating to smoking habit, employment status, and household income.

The data concerning the asthma severity measures are shown in table 1. The overall severity score ranged from 2 to 24 with a mean (SD) of 11.3 (5.1). All patients experienced symptoms of wheeze, chest tightness, cough, or shortness of breath on exercise. Only two did not require to use an inhaled bronchodilator, at least on an occasional basis, and 92 (90%) were taking prophylactic therapy in the form of inhaled corticosteroids or sodium cromoglycate. The FEV₁ was more than 80% of predicted normal values in 57 (60%) of patients; 85% of patients were atopic with one or more positive skin prick tests to common allergens.

HOUSING CONDITIONS SURVEY

Dampness surveys were conducted for 75% of the sample. A comparison between those surveyed (n = 222) and those not surveyed (n = 76) showed no difference in sociodemographic characteristics including subject sex, age, household size, housing tenure, duration of tenancy, weekly household income, cigarette smoking, pet ownership, or self-reported dampness. The only significant difference concerned employment where fewer respondents in the surveyed households were employed (p = 0.01).

One hundred and ten (49%) of the dwellings surveyed were categorised as dry. Excess moisture was detected in 112 homes (51%) with 43 (19%) containing at least one area of grade 3 or 4 (severe) dampness. Mould growth was observed in a total of 57 dwellings (26%) and in 33 homes (15%) was classified as significant (total mould score ≥ 3). There was a strong relationship between the presence of dampness and mould within a dwelling. Forty nine (86%) dwellings with evidence of visible mould growth also had areas of detectable dampness and there was a significant correlation between the total mould and total dampness scores of a dwelling (r = 0.51, p = 0.0005).

There was a tendency for both asthmatic and control subjects to underestimate dampness in the home. Dampness was detected in the dwellings of 21 (52%) asthmatic and 27 (32%) control subjects who claimed their homes were dry. Agreement between self-reported dampness and the findings of the surveyor occurred in 83 homes (63%) of control subjects and 56 (63%) of asthmatic subjects.

Table 3 Prevalence of dampness in dwellings of asthmatic and control subjects

Dampness measures	Controls	Asthmatic subjects*			χ^2 trend	p value
		Mild	Moderate	Severe		
Self reported	(n = 196)	(n = 33)	(n = 32)	(n = 32)		
Any dampness/condensation	76 (39)	16 (48)	18 (56)	20 (62)	8.72	0.003
Serious dampness/condensation	22 (11)	6 (18)	12 (37)	15 (47)	30.5	0.00005
Previous house damp	56 (28)	12 (36)	16 (50)	17 (53)	11.3	0.0008
Moved because damp house	20 (10)	5 (15)	8 (25)	8 (25)	8.33	0.004
Observed	(n = 132)	(n = 29)	(n = 29)	(n = 27)		
Any dampness	54 (41)	13 (45)	20 (69)	21 (78)	16.2	0.00006
Severe dampness ¹	19 (14)	4 (14)	8 (28)	10 (37)	8.20	0.004
Any mould	30 (23)	5 (17)	8 (28)	12 (44)	4.24	0.04
Significant mould ²	15 (11)	3 (10)	6 (21)	9 (33)	8.10	0.005

* Severity score not available in five cases due to incomplete data.

¹ Dwellings in which at least one dampness measurement was of grade 3 or 4 severity.

² Dwellings with total mould score ≥ 3 .

Figures represent numbers (percentages).

Table 4 Crude and adjusted odds ratios for asthmatic subjects living in dwellings with evidence of dampness

Dampness measures	Crude OR (95% CI)	Adjusted OR* (95% CI)
Self reported	(n = 298)	(n = 283)
Any dampness/condensation	1.92 (1.18 to 3.12)	1.93 (1.14 to 3.28)
Serious dampness/condensation	4.13 (2.26 to 7.55)	5.45 (2.81 to 10.6)
Previous home damp	2.11 (1.29 to 3.47)	2.55 (1.49 to 4.37)
Moved because previous home damp	2.28 (1.17 to 4.44)	2.08 (1.02 to 4.24)
Observed	(n = 222)	(n = 211)
Any dampness	2.62 (1.55 to 4.72)	3.03 (1.65 to 5.57)
Severe dampness ¹	2.14 (1.06 to 4.03)	2.36 (1.34 to 4.01)
Any mould	1.46 (0.82 to 2.78)	1.35 (0.79 to 2.28)
Significant mould ²	2.23 (1.21 to 5.32)	1.70 (0.78 to 3.71)

¹Dwellings in which at least one dampness measurement was of grade 3 or 4 severity.

²Dwellings with total mould score ≥ 3 .

* Adjusted for subject age, sex, weekly household income $<£200$, unemployment, respondent smoker, other smoker in household and pet ownership. Fifteen subjects did not disclose household income and were not included in the logistic regression models. Odds ratios and 95% confidence intervals calculated from logistic regression coefficients.

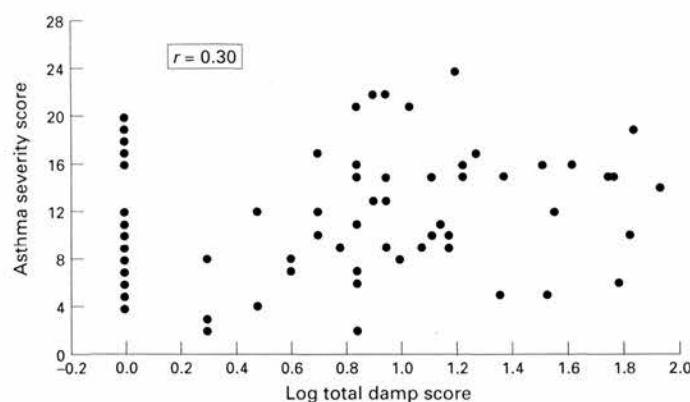


Figure 1 Scatter plot showing the relationship between severity of asthma and severity of dampness in the dwellings.

The frequencies of both self-reported and observed dampness and mould in the dwellings of asthmatic and control subjects are listed in table 3. There were significant trends for the prevalence of both measures of dampness or mould to rise with increasing severity of asthma.

The mean indoor temperature in the homes of asthmatic subjects was slightly lower than that in the homes of control subjects (16.7°C versus 17.7°C, $p=0.023$). Although homes without central heating were slightly colder than those with central heating (mean indoor temperature 16.7°C versus 17.7°C, $p=0.006$), there was no significant difference in the use of this mode of heating in homes of asthmatic and control subjects ($p=0.4$). No significant difference was observed in the mean indoor relative humidity measurements from the homes of asthmatic and control subjects (57% versus 56%, respectively, $p=0.5$).

CONTROLLING FOR CONFOUNDING VARIABLES

Possible confounding variables identified using univariate statistics included employment status, household income, respondent smoking, and the presence of another smoker in the household. To control for the possible confounding effect of these variables, as well as age, sex and pet ownership, logistic regression models were constructed with asthma as the dependent variable and the measures of damp-

Table 5 Multiple regression analysis

Asthma severity measure	B (95% CI)	p value
Asthma severity score	2.30 (0.53 to 4.07)	0.01
FEV ₁ (% predicted)	-11.9 (-19.4 to -4.39)	0.03
FEV ₁ /FVC ratio	-5.79 (-10.2 to -1.35)	0.01

B = regression coefficient; FEV₁ = forced expiratory volume in one second; FVC = forced vital capacity.

Associations between measures of asthma severity and total dampness scores, controlling for unemployment, household income (above/below £200), respondent smoker, other smoker in house, and pet ownership.

ness or mould and the confounding variables entered as covariates. The crude and adjusted odds ratios for self-reported and surveyor observed damp are listed in table 4. The adjusted odds ratios for the various measures of damp in the dwelling differed little from the crude ratios, suggesting that the above variables had only a small confounding effect. However, the adjusted odds ratios for the presence of mould in the dwelling were lower than the crude ratios with 95% confidence intervals that included unity.

DAMP HOUSING AND ASTHMA SEVERITY

The relationship between asthma severity and increasing severity of dampness or mould in the dwelling was assessed in the asthmatic subjects. The total dampness scores ranged from 0 to 85 with a median of 6. Total mould scores were likewise positively skewed and logarithmic transformations were used to normalise the distributions. The relationship between asthma severity and the severity of dampness in the dwelling is shown in fig 1. A statistically significant positive correlation was seen between asthma severity and total dampness scores ($r=0.30$, $p=0.006$). A similar significant correlation was also observed between the asthma severity and total mould scores ($r=0.23$, $p=0.035$). The greater the severity of dampness or mould in the home, the more likely the patient was to have more severe asthma.

DAMP HOUSING AND LUNG FUNCTION

There were significant negative correlations between the total dampness score for the dwelling and the percentage predicted FEV₁ ($r=-0.30$, $p=0.006$) and FEV₁/FVC ratio ($r=-0.25$, $p=0.023$). These correlations remained statistically significant after controlling for the confounding factors mentioned previously (table 5). Patients living in homes in which the surveyor had confirmed evidence of dampness had a lower FEV₁ (mean difference 10.6%, 95% CI 1.0 to 20.3) and FEV₁/FVC ratios (mean difference 5.4%, 95% CI -0.1 to 10.9) than those living in dry dwellings. Overall, the severity of dampness within the dwelling accounted for approximately 7% of the variance in FEV₁.

Discussion

The results of this study show that asthmatic patients attending a hospital asthma clinic were two to three times more likely to live in a

dwelling with evidence of dampness than an age and sex matched random sample of the general population living in the same area of the city of Glasgow. There were significant trends for the prevalence of both self-reported and observed measures of dampness to rise with increasing severity of asthma. Furthermore, there were significant correlations between asthma severity and, in particular, severity of airflow obstruction and the severity of dampness and mould in the dwelling, suggestive of a dose-response relationship.

Most previous studies investigating the effects of damp housing on health have relied on the same questionnaire to elicit information on both the subject's health and indicators of dampness in the dwelling, raising the possibility of respondent bias.^{21,23} Although our asthmatic subjects reported more dampness, condensation, and mould in both their current and previous dwellings than did control subjects, they were unaware of the purpose of the study at the time of completion of the questionnaire. Furthermore, we adopted an independent, standardised, semiquantitative approach to the assessment of dampness in each dwelling to avoid having to rely solely on measures of self-reported dampness. It is of interest that both asthmatic and control subjects under-reported the presence of dampness in the home to the same degree, suggesting that asthmatic subjects do not over-report dampness in the home and significant respondent bias was unlikely to have occurred. As dampness and mould in the dwelling was measured objectively and the diagnosis of asthma had been previously established by a respiratory physician, investigator bias was also effectively eliminated.

Asthmatic subjects were more likely to be unemployed or receiving invalidity benefit and therefore live in a household with a lower net income. This could result in their gravitating towards poorer quality housing which could be more prone to dampness and more difficult to heat. However, most of our asthmatic and control subjects lived in local authority housing and it is unlikely that asthmatic subjects were preferentially allocated poorer housing. Furthermore, the association between asthma and damp housing remained statistically significant after controlling for these confounding variables.

We are not currently aware of any previous study that has identified a dose-response relationship between damp housing and asthma severity. This study identified significant trends for higher prevalences of dampness and mould in the dwellings of subjects with increasing severity of asthma, and statistically significant correlations between asthma severity and quantity of dampness and mould in the home. These findings strengthen the case for a dose-response relationship.

Our reported odds ratios for asthmatic subjects living in damp homes are in keeping with those described in previous studies where associations were reported between respiratory symptoms such as wheeze and cough and dampness or mould in the home,^{7,8,13} but they are higher than those previously quoted for

associations between asthma and dampness in the home. The trend for a rise in the prevalence of dampness in the home with increasing severity of asthma (table 3) suggests that the higher odds ratios reported in this study are likely to be due to our selection of a high proportion of patients with moderate to severe asthma.

There are several plausible biological mechanisms to explain a higher prevalence of asthma in subjects living in damp dwellings. The house dust mite *Dermatophagoides pteronyssinus* is known to thrive in damp conditions and both mite numbers and allergen levels have been shown to increase with both higher indoor humidity^{4,18} and indicators of dampness in the home.^{4,25} Measurements of exposure to HDM were not undertaken in this study but it has been shown that increased exposure to HDM allergen may result in increased sensitisation²⁶ and act as an exacerbating factor for asthma.²⁷ Storage mites have also been found in significant numbers in house dust from damp dwellings.^{28,29} Allergy to these mites has been implicated as a cause of occupational asthma in farmers³⁰ but it is not clear whether this allergy is present in subjects who live in damp dwellings but have no relevant occupational exposure.

The presence of mould growth in dwellings in this study was much more common in those which also had areas of severe dampness. The self-reporting of mould in a dwelling, as used in previous studies, may therefore act only as a marker for the presence of severe dampness and hence higher exposure to HDM allergens. Alternatively, exposure to moulds and airborne fungal spores can exacerbate respiratory symptoms in susceptible individuals.³¹ It is estimated that 10–15% of asthmatic subjects have allergy to moulds, assessed by skin prick testing, most commonly to *Aspergillus fumigatus*, *Alternaria alternata*, *Penicillium*, and *Cladosporium*.^{32–34} However, they frequently also have allergy to other antigens such as the house dust mite and the contribution of the mould allergy to their disease is often difficult to assess.

It is generally agreed that both the prevalence and severity of asthma are increasing^{35–37} and more attention is being focused on possible environmental factors which may account for this. We have shown that there is an association between asthma and damp housing but our asthmatic subjects were selected from a hospital outpatient clinic and are likely to represent the more severe end of the disease severity spectrum. Whether dampness in the home predisposes to the development of asthma or aggravates the severity of the disease clearly requires further investigation, but the results of this study suggest that action to reduce dampness in the home could favourably influence asthma morbidity.

This study was supported by a grant from the Chest, Heart and Stroke Association, Scotland.

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